# THE PLANT DISEASE REPORTER

Issued By

## THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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# A PRELIMINARY SURVEY OF PLANT DISEASES IN GUATEMALA

Albert S. Muller

Included in the Mycological collections made by the writer and maintained at the National School of Agriculture, may be found material which serves as a basis for the presentation of the following preliminary survey of the plant diseases of Guatemala. Most of the diseases have been the object of observation during the period 1941 to 1950. Sixty host plants are included in the study.

Bardales (1) in 1928 described three coffee diseases found in Guatemala. Palm (2) in 1932 published a short list of plant diseases on a dozen cultivated plants in Guatemala. Alvarado (3) in 1933, in his treatise on coffee production, gives accounts of more than two dozen coffee diseases, of which not more than a few have been recognized by other workers, as of consequence.

In 1944, Stevenson and Wellman (4) published an account of plant diseases in El Salvador, a country neighboring on Guatemala, of similar crops, topography and plant disease problems which Wellman discusses (5) in articles published in 1946.

## GARDEN CROP DISEASES

**ALLIUM SPP.**: Onions are usually subject to damage by Alternaria porri, and frequently leek also.

**APIUM GRAVEOLENS**: Celery suffers damage on older foliage by Septoria apii.

**BETA SPP.**: Beets and mangles are usually affected by Cercospora beticola, which damages swiss-chard even more.

**BRASSICA SPP.**: Cabbage production is less affected by Alternaria brassicae than cauliflower. Seedbeds are sometimes destroyed by Peronospora parasitica.

**CAPSICUM SPP.**: Peppers and chiles are frequently defoliated by Cercospora capsici and sometimes black mold, Cercospora diffusa is found. Mosaic when present is severe. Rust, caused by Puccinia paulensis is infrequent.

**CITRULLUS VULGARIS**: Watermelons have a short production period due to defoliation by Pseudoperonospora cubensis.

**CUCUMIS SATIVUS**: Cucumber production is always seriously affected by Pseudoperonospora cubensis. Powdery mildew, Erysiphe cichoracearum, is usually present. Mosaic (virus) when present is very severe.

**CUCURBITA SPP.**: Squash and pumpkins usually produce well in spite of Pseudoperonospora cubensis and Erysiphe cichoracearum. Most named imported squashes suffer from a severe type of mosaic, while local unnamed squashes do not.

**DAUCUS CAROTA**: Carrot plantings sometimes fail completely due to Alternaria carotae, which blights the foliage.

**LACTUCA SATIVA**: Lettuce grown in the rainy season has been found to be severely damaged by Bremia lactucae and by Sclerotinia sclerotiorum. Older foliage is usually damaged by Septoria lactucae.

**LYCOPERSICUM ESCULENTUM**: Tomato production in the rainy season generally fails due to Phytophthora infestans, but usually this disease is absent in the dry season, November to May. The production period may be shortened by Alternaria solani and Cladosporium fulvum in the dry season and as a result of the root nematode, Heterodera marioni (Meloidogyne sp.). Leaf spot caused by Septoria lycopersici is never severe. Mosaic (virus) may be severe.

**PISUM SATIVUM**: Peas usually have a shortened production period due to Erysiphe polygoni. Pods have been found to be seriously damaged by Mycosphaerella pinodes, and less frequently by Colletotrichum pisi.

**RAPHANUS SATIVUS**: Radishes are affected by Alternaria brassicae and Albugo candida, but not seriously.

**SOLANUM MELONGENA**: Eggplants frequently suffer from defoliation and fruit rot due to Phomopsis vexans.

**TRAGOPOGON PORRIFOLIUS**: Salsify, little planted, suffers from white rust, Albugo trapopogonis.

**VICIA FABA**: Broad beans are always subject to severe defoliation at maturity by Uromyces fabae, sometimes causing failure.

## FIELD CROP DISEASES

ARACHIS HYPOGAEA: Peanut foliage at maturity is always affected by Cercospora personata, but serious crop losses are only occasional. Wilt caused by Sclerotium rolfsii has been found in wet periods and rust, Puccinia arachidis, in seasons that are unusually dry.

AVENA SATIVA: Oats are very severely attacked in some years by Puccinia coronata and less frequently by Puccinia graminis var. avenae in only occasional fields. Smut caused by Ustilago avenae is usually present but affecting a minimum number of plants.

CAJANUS INDICUS: Pidgeon peas lose mature foliage either from Cercospora cajani or Uromyces dolicholi without seriously damaging the crop.

EUCHLAENA MEXICANA: Teosinte for forage suffers from rust, Puccinia sorghi and from stripe, Helminthosporium turcicum. Smut, Ustilago zeae, is occasional.

GLYCINE SOJA: Soybeans are affected near maturity by two leafspots, one caused by Cercospora sojina and the other by Mycosphaerella pinodes. In wet seasons wilt is sometimes caused by Sclerotium rolfsii and pod rot, caused by Corticium vagum produces much loss of seed.

GOSSYPIUM SPP: Cotton is not seriously affected by any disease, but rust, Kuhneola gossypii is usually present on mature foliage and a leaf spot caused by Alternaria gossypii.

HORDEUM VULGARE: Barley is very severely damaged every year by Helminthosporium sativum, and no variety yet tested has shown any resistance. Smut, Ustilago hordei attacks a minimum of plants in occasional fields. Rust, Puccinia simplex (P. hordei), has been found in only one field.

IPOMOEA BATATAS: Sweet potatoes are affected without serious damage by Phyllosticta batatas and by Albugo ipomoeae-panduranae. Losses in storage are often severe due to Rhizopus nigricans.

MANIHOT UTILISSIMA: Cassava foliage usually is affected when mature by Cercospora henningsii without damage, and the root crop damaged when poorly handled in storage by Rhizopus nigricans.

MEDICAGO SATIVA: Alfalfa is severely damaged in dry periods by rust, Uromyces medicaginis, while following rainy periods loss of foliage is caused by Pseudopeziza medicaginis in most fields and by Cercospora medicaginis in some fields.

NICOTIANA TABACUM: Tobacco foliage when mature is attacked by Cercospora nicotianae without severe losses. Mosaic (virus) is very severe some years in some fields.

ORYZA SATIVA: Rice foliage when mature is attacked by Cercospora oryzae without much crop loss. In some years Piricularia oryzae affects young plants severely and later causes losses by neck-rot. Seedling blight caused by Helminthosporium oryzae sometimes reduces stands greatly.

PHASEOLUS SPP.: Bean rust, Uromyces phaseoli var. typica, causes poor crops in some years and also anthracnose, Colletotrichum lindemuthianum, while in one year beans in one locality produced poorly due to Septoria blight. Cercospora columnare is frequently found doing some damage and also Erysiphe polygoni. Crop failure in one locality was due to mosaic (virus) which is present every year, but usually not apparent on the majority of the plants.

SACCHARUM OFFICINARUM: Sugar cane is never without spotting by Leptosphaeria sacchari causing very little damage. Only one field has been found damaged by the leaf spot caused by Helminthosporium sacchari. Mosaic is now of no importance due to the use of P.O.J. varieties which are immune.

SESAMUM INDICUM: Sesame plants under poor growing conditions are severely blighted by Alternaria solani. Mature plants are affected by leaf spot, Cercospora sesami, which in some fields reduces yield by severe early defoliation.

SOLANUM TUBEROSUM: Potatoes grown in the rainy season are subject to almost total loss due to Phytophthora infestans, which attacks many indigenous varieties as well as imported varieties. Early blight, Alternaria solani has not caused serious crop reduction. Crop failures due to leaf roll have been observed but never by mild or rugose mosaic which occur sporadically. Bags of tubers in markets often show considerable scab, Actinomyces scabies and black sclerotia of Rhizoctonia solani, but no economic losses are recorded. Root nematode, Heterodera marioni (Meloidogyne), has destroyed some fields.

SORGHUM VULGARE: Sorghums, grain and forage are sometimes severely damaged by rust, Puccinia purpurea, which is always present. Sorghum relatives such as Sudan and Johnson grass often fail as forage crops due to this rust. Sorghums also suffer severe foliage blight due to Helminthosporium turcicum. Grain sorghums maturing in rainy periods are severely affected by scab, Gibberella saubinetii (G. zeae) and some imported varieties show considerable smut, Sphacelotheca sorghi.

TRITICUM SPP.: Wheat fields, usually mixed varieties, annually are defoliated while the heads are still green by leaf rust, Puccinia rubigo-vera, while stem rust, Puccinia graminis var. tritici is sporadic, entirely absent some years, or when present, severe or benign, seemingly depending on date of planting and growth conditions, some fields maturing before damage is great. Glume rust, Puccinia glumarum has been found in only one field. A little smut, Ustilago tritici is usually found but the disease is no consequence. Two leaf spot diseases, causing little damage are found, due to Septoria tritici and Helminthosporium sativum.

VIGNA SINENSIS: Cowpeas are subject, depending on weather conditions, to defoliation by Mycosphaerella pinodes and to leaf spot caused by Cercospora cruenta. Mosaic is sometimes very severe on some varieties.

ZEA MAYS: Corn production is reduced in some years by stripe, Helminthosporium turcicum, and by ordinary rust, Puccinia sorghi, but usually these two diseases are not so serious. Mature foliage is frequently affected by Cercospora zeae-maydis and by Phyllachora maydis, and leaf sheaths by Physoderma zeae-maydis, without serious damage. Smut, caused by Sorosporium reilianum, when it occurs, is serious, while ordinary smut, Ustilago zeae, is never very damaging. In some years dry rot, Diplodia zeae is very serious but usually not alarming, and only in one year was cob rot, Nigrospora sphaerica a serious problem. Pink rot, Fusarium moniliforme is of common occurrence following earworm injury. A virus disease designated as stunt seems to be increasing in importance.

## FRUIT DISEASES

CARICA PAPAYA: Papaya foliage is often attacked without serious damage by Pucciniopsis caricae, and by Oidium caricae. Fruit in storage rots quickly when attacked by Colletotrichum papayae. Mosaic is not yet widespread but is very destructive when present.

CITRUS SPP.: Citrus gummosis has been found to be caused by Phytophthora parasitica, and it is not a major disease. Dieback of twigs occurs infrequently, caused by Colletotrichum gloeosporioides. Sour orange and lemons suffer foliage and fruit damage, due to Elsinoe fawcettii. Weak trees suffer foliage and twig damage due to Alternaria citri. Fruit spoilage has been found due to Penicillium digitatum, Diplodia natalensis, and Rhizopus nigricans. Felt disease is the result of Septobasidium pseudopedicellatum.

COFFEA ARABICA: Severe defoliation and some berry spot is found in limited areas, caused by Omphalia flavida, but over wide areas the disease is not serious, as is the case of thread blight, Corticium koleroga. Leaf spot, Cercospora coffeicola, damages severely poorly shaded nursery stock and together with Colletotrichum gloeosporioides causes dieback, leaf and berry spot on older trees in areas deprived of sufficient shade. In cases of root rot the presence of Rosellinia spp. in or on affected plant tissue has usually been observed, and the disease is frequently serious in limited areas.

CYDONIA OBLONGA: Quince leaf spot is common, caused by Cercospora mali,and fruit rot, due to Monilinia cydoniae,is sometimes found in the Monilia stage.

FICUS CARICAE: Fig rust, Cerotelium fici is common and the cause of reduced production.

FRAGARIA CHILOENSIS: Strawberry leaf spot, Ramularia fragariae is common, and cases of severe damage by fruit rot, Botrytis cinerea have been found.

MANGIFERA INDICA: Mango inflorescences are greatly damaged by Colletotrichum gloeosporioides, which also causes fruit drop on a large scale and in some years considerable dieback and foliage blight. Oidium mangiferae sometimes affects new flushes of foliage seriously and drop of small fruit is the result.

MUSA SPP.: Banana production is impossible without control measures against Sigatoka, caused by Cercospora musae. Increasingly large areas are being abandoned because of Panama disease, Fusarium oxysporum. var. cubense. Oval leaf spot caused by Cordana musae is common but not serious. Fruit rot, Gloeosporium musarum, destroys much locally stored ripe fruit.

PERSEA AMERICANA: Avocado scab, Sphaceloma perseae is often serious on nursery stock, but not important in crop production. Phyllachora gratissima is also not important. Anthracnose sometimes reduces bearing by attacking inflorescences and small fruit which drop, and is due to Colletotrichum gloeosporioides.

PUNICA GRANATUM: Pomegranate leaf spot, Cercospora punicae is common but not serious.

PRUNUS PERSICA: Peach leaf rust, Tranzschelia pruni-spinosae, is very common and may be more serious than supposed. Sclerotinia cinerea causes some fruit loss.

PYRUS SPP.: Apple canker, Sphaeropsis malorum has been found in serious proportions in a few orchards and in the same orchards scab, Venturia inaequalis has been common. Leaf spot, Cercospora mali is common but not serious.

THEOBROMA CACAO: Cacao fruit rot, Phytophthora palmivora is seasonal, causing much loss certain years and others very little. Pod rot, Diplodia cacaoicola is not a major problem, nor is spotting by Colletotrichum gloeosporioides, which sometimes, however, affects many small fruits which are lost.

VITIS SPP.: Grape production is limited by downy mildew Plasmopara viticola, which is usually very severe and by powdery mildew, Oidium tuckeri. Anthracnose, Sphaceloma ampelinum seriously affects the fruits of some varieties as does rust, Phakopsora vitis, the foliage of others.

## FLOWER DISEASES

ANTIRRHINUM MAJUS: Snapdragon rust, Puccinia antirrhini, is very severe when it occurs. A severe leaf spot has been attributed to a new and unpublished species of Cercospora.

CANNA INDICA: Cannas are commonly affected without great damage by Puccinia cannae.

DAHLIA SPP.: Dahlias, both cultivated and wild show considerable leaf spotting on mature foliage due to Cercospora grandissima and after flowering are damaged by Oidium sp. Mosaic occurs sometimes.

DIANTHUS CARYOPHYLLUS: Carnations are affected by rust, Uromyces caryophyllinus and leaf spot, Heterosporium echinulatum.

IMPATIENS BALSAMINA: Balsams are usually heavily attacked after flowering by Oidium sp.

JASMINUM OFFICINALE: Jasmine foliage is sometimes heavily attacked by Cercospora jasminicola.

PETUNIA HYBRIDA: Petunia foliage is often heavily spotted by Cercospora petuniae, and the flowers rotted in wet spells by Botrytis cinerea.

ROSA SP.: Roses are commonly affected by Cercospora rosaecola and by Oidium leucononium. Scab, Sphaceloma rosarum, is less frequent as is black spot, Actinonema rosae. Rust, Phragmidium disciflorum defoliates some varieties.

STATICE ARMERIA: Statice or thrifts are commonly affected after flowering by Cercospora staticis.

ZINNIA ELEGANS: Zinina foliage is commonly affected by Cercospora atracinta and by Odium sp.

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ESCUELA NACIONAL DE AGRICULTURA, FINCA "BARCENA" - VILLA NUEVA, GUATEMALA, C. A.

# OBSERVATIONS ON MICROBIOLOGICAL DETERIORATION OF COTTON FIBER DURING THE PERIOD OF BOLL OPENING IN 1949

P. B. Marsh[1], L. R. Guthrie[2], K. Bollenbacher[3], and D. C. Harrell[4]

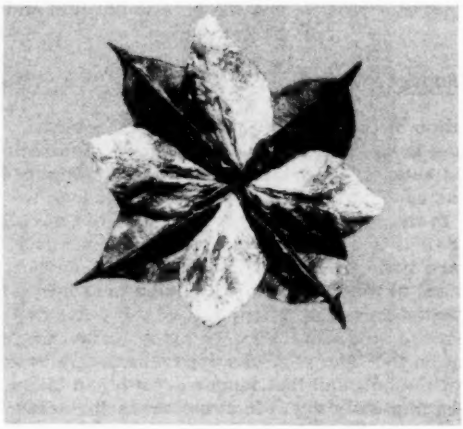

Figure 1. Cotton boll infected with Colletotrichum gossypii and exhibiting anthracnose tight lock. Infected bolls are usually, but not invariably, symmetrical in appearance and the locks are free rather than bound to the carpel wall by fungus mycelium.

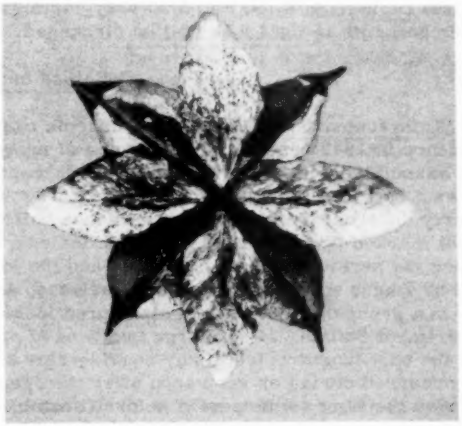

Figure 2. Cotton boll infected with Diplodia sp. and exhibiting Diplodia tight lock. Infected bolls are usually but not invariably symmetrical in appearance. The growth is darker than in the case of anthracnose tight lock and is speckled, due to agglomeration of the pycnidia in clumps.

Microbiological fiber deterioration at the time of boll opening is one of the more important problems in the growing of cotton in areas where wet weather occurs frequently at this period. Few observations and experimental records, however, appear to have been reported on the subject. The amount of damage which occurs is very much dependent on the weather over a period of a few days at the time of opening of the bolls and consequently is extremely variable from one season to the next in any locality. In the vicinity of Florence, South Carolina, a very considerable amount of damage of this nature occurred in 1949. The present paper is a report of certain observations on the difficulty as it was observed at and in the vicinity of the Pee Dee Experiment Station at Florence and of certain subsequent observations on collected material made at Beltsville, Maryland. The results of a survey by Dr. C. H. Arndt of similar fungus damage over a large part of South Carolina during the same period (3) are presented in an accompanying article.

An almost continuous succession of cloudy and rainy days occurred at Florence during the three-week period starting on August 15. Much of the cotton in the area came to the stage of opening of the bolls during this time interval and many of these bolls suffered fungus attack on the fiber. The affected locks[5] showed partial or complete failure to fluff, were slightly to severely discolored with fungus growth, and contained fiber which was obviously much weakened in many cases. This symptom complex has been referred to in a previous paper (14) as "tight lock" and was shown in earlier observations (1, 14) to be associated with fungi of the genera Alternaria, Cladosporium, Diplodia, and Fusarium, and with Colletotrichum gossypii (imperfect form of

Note: Thanks are due to Mr. E. E. Hall, Superintendent of the Pee Dee Station, and to Mr. W. H. Jenkins of the same station for many courtesies during the course of this work, also to J. B. Dick, D. C. Neal, A. L. Smith, and J. H. Turner for cotton samples.

1, 2, and 3 Physiologists and botanist respectively, Division of Cotton and Other Fiber Crops and Diseases, U. S. Department of Agriculture, Beltsville, Maryland.

[4]Agronomist, Pee Dee Experiment Station, Florence, South Carolina.

[5]The term "lock" is used here to refer to the lint and seed within a single locule.

Glomerella gossypii), the cotton anthracnose fungus. These fungi are all known to be cellulose-decomposing organisms and to be capable of causing loss in strength of cotton fiber (15). In contrast to the problem here described, growth of micro-organisms on the fiber during prolonged weathering after it has once dried and fluffed in the normal manner produces a dull graying of the fiber, sometimes called "blue stain", which is distinct from tight lock and which is associated with certain micro-organisms identified in a previous paper (14). In addition to tight locks, the typical Diplodia boll rot was also very common at Florence in 1949 and the relation of this and other boll rots to tight lock will be discussed.

## Nature of the Damage

Anthracnose Tight Lock. Microscopic examination of discolored tight locks collected at Florence in 1949 resulted in the finding of many cases in which a major part of the discoloration was associated with the presence of the dark-colored setae of the anthracnose fungus. Figure 1 shows a boll affected with this type of tight lock. The appearance of anthracnose tight lock is clearly different from that of anthracnose boll rot. In the former, the outer surface of the boll is not marked by conspicuous necrotic lesions, the boll is not grossly distorted by fungus attack during the period of its enlargement, and the locks are little if at all bound to the inner carpel wall by fungus mycelium. In the latter case, any or all of these symptoms may be present (28).

In a group of 38 bolls with discolored locks collected from the Keitt plots on the Pee Dee Experiment Station, 24 had large numbers of anthracnose setae on them. In many instances this was the only fungus noted in any considerable amount on the fiber. It was apparent that infection had occurred during or very soon after the opening of the boll and that fungus growth had taken place on the fiber in the period before it had become completely dry. In some cases the setae occurred over essentially the entire lock surface, while in others only a small part of the surface was concerned. All degrees of variation between these two extremes occurred, with the amount of fungus growth and the degree of compactness of the lock apparently being related to the length of the period of drying of the fiber. The setae of the fungus also were found in many cases growing upon and causing a darkening of the endocarp or inner wall of the burr.

As a check on the identity of the fungus, ten isolations were made from discolored locks and ten from discolored inner boll walls, inoculum of each isolate was added to moist sand in a moist chamber, and cotton seeds then planted in the sand. Each isolate produced the usual killing of seedlings and the severe necrotic lesions characteristic of anthracnose seedling infection. The seed in the control germination dishes, to which sterile water rather than fungus inoculum had been added, exhibited 90 to 100 percent germination and normal seedling growth. In addition to the dark-walled, spore-bearing setae of anthracnose tight locks, the fungus also commonly produces hyaline conidia on the fiber among the setae. Examination of the outer boll surfaces indicated that the setae were frequently present in abundance on the outer surface of the boll even when macroscopic observation showed no apparent lesions, also when no setae could be found on the fiber by microscopic examination. The setae themselves are capable of germination on agar, Figure 3, and are presumably able also to infect seedlings.

In some bolls, only one or two of the locks were attacked by the fungus. This situation, which has been pictured before (1, p. 937), occurred particularly in the case of horizontally-oriented bolls. It was obviously related to the fact that dehiscence is initiated on the upper side of such bolls and that inoculation with the fungus may take place onto the exposed but still undried fiber of only one or two locks.

Among the early observations of particular pertinence to our own findings on anthracnose tight lock at Florence was a report by Barre in 1909 (5) of the occurrence of the anthracnose fungus on cotton fiber in the boll and his discussion of the probable importance of the fungus there as a source of seedling inoculum. The most pertinent passages from this paper appear to be the following: "In some cases no sign of anthracnose would be apparent on the surface of the bolls, and the lint and seed would be mature and the bolls open, the only indication of disease being a slight discoloration and slight compactness of the lint. Upon examination of such bolls, numerous anthracnose spores would be found in the discolored patches of lint...." and, in respect to seed from such locks, "it seems ...that this is about the kind of seed that is most effective in spreading the disease". Although this original description by Barre and an accompanying illustration clearly relate to a mild form of the symptom complex observed by us at Florence in 1949 in connection with the growth of C. gossypii on the fiber and here designated as "anthracnose tight lock", except for a passing reference by Edgerton (9) in 1912 no record has been found to indicate that it has been observed again in the intervening years. This circumstance may possibly be related in part to the fact that, in spite of the well-known early contributions of Southworth (25),

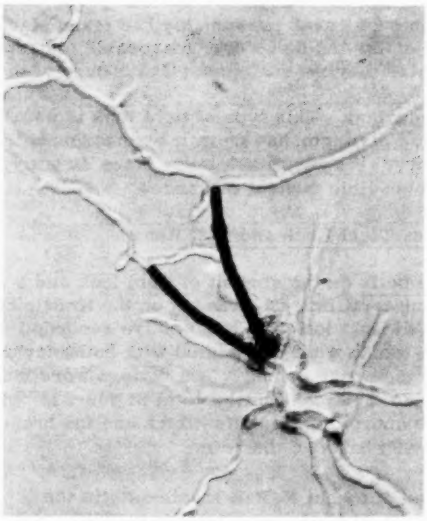

Figure 3. Setae of the anthracnose fungus, Colletotrichum gossypii, germinating on the surface of cotton extract-mineral salts agar.

Atkinson (4), and Barre (5, 6) on cotton anthracnose, some doubt existed in later years in regard to the importance of the cotton anthracnose fungus as a seedling pathogen, until the point was firmly re-established by recent studies (17, 19, 32). Miller (19) stated in 1943, "Previous to our surveys, it was generally assumed that anthracnose practically disappeared with the advent of the boll weevil...".

In respect to the possible significance of anthracnose tight locks as a source of seedling inoculum, it is to be noted that Weindling and Miller (30) and Miller (18) in 1943 emphasized the importance of contamination of healthy seed at the gin with trash derived from diseased seed cotton, i.e. (30) with "broken bolls, hulls, pieces of leaves, stems, bracts, petioles, and other debris that has been picked with the seed cotton". Anthracnose tight locks obviously could also be very important as a source of inoculum which could be spread at the gin.

In addition to Colletotrichum tight locks, tight locks from the Keitt plots were found which were infected with Fusarium, Alternaria, Curvularia, Nigrospora, and Rhinotrichum.

Diplodia Tight Lock and Boll Rot. Diplodia boll rot has been reported to be of common occurrence in Florida (28), Arkansas (10), Mississippi (20), and Louisiana (9, 21). The fungus is usually regarded as a wound parasite (9, 21, 28) and as a generalized type of pathogen capable of cross-infection among many genera and species of host plants (27). On cotton, it produces a characteristic boll rot which has been known for many years and which is distinguished by the presence of the black pycnidia of the fungus and by the two-celled spores which occur in sooty-black profusion over the surface of the boll. The fungus was found associated at Florence in 1949 not only with the typical Diplodia boll rot but also with a symptom complex which might be described as Diplodia tight lock (Fig. 2), in which the boll opened partially or often completely, and compact, very dark-colored locks were found. A complete range of symptom complexes--intermediate in appearance between tight lock and typical Diplodia boll rot (28)--was also noted.

In accord with previous observations (10, 21), typical Diplodia boll rot as seen at Florence was very frequently associated with a considerable amount of decomposition of the tissues at the base of the boll, so that, when the boll was picked, it had a decided tendency to break off at the point where the pedicel joins the boll proper rather than at the normal position at the base of the pedicel. It would appear that infection leading to Diplodia boll rot frequently takes place at the base of the boll. The fungus then progresses upward through the vascular tissue and throughout the endocarp, which frequently becomes brown before damage is obvious externally. It then grows outward into the outer wall of the carpel, which eventually becomes almost cheesy in consistency, and inward throughout the fiber. A boll in which such a sequence of events has taken place never opens and is completely worthless. Under humid conditions, the pycnidia push out sooty masses of dark two-celled spores (14), over the outside of the boll. Hyaline, one-celled, immature spores of the same shape are also frequently found.

In a fairly considerable number of cases of Diplodia boll rot, the characteristic brownish discoloration and later black-pin-head pycnidial formation was seen to occur first adjacent to a

split at a line of dehiscence in the boll wall. Infection in such cases presumably had taken place at a time when weather following the first cracking was moist and therefore unfavorable for rapid drying and opening of the bolls. This type of infection also may result in complete failure of the boll to open and in destruction of its contents.

At Florence in 1949 Diplodia tight lock was very prevalent. This type of tight lock is usually darker than anthracnose tight lock and frequently may be distinguished from it by macroscopic observation from the appearance of the pycnidia in clumps. Fungus attack takes place directly upon the fiber, an important consideration in respect to possible control measures.

## Factors Influencing the Incidence of Fungus Tight Lock and Boll Rot

The effect of the density of leaf covering around the bolls on the amount of tight lock and boll rot in cotton was clearly seen in the plants of a long-term fertilizer experiment on the Keitt plots at the Pee Dee Experiment Station. Ratings for amount of tight lock and boll rot are recorded in Table 1 for the 4-8-4, 6-8-4, and 10-8-4 treatments, which were associated with both increasing leafiness and increasing amounts of fiber damage in the order named. The ratings were made on all bolls of single plants selected at random within each treatment until a total of 200-225 bolls had been rated. In the 10-8-4 treatment the plants were approximately 5 feet high and the branches of plants in adjacent rows interlaced, shading the ground between the rows.

Table 1. Amount of fungus tight lock and boll rot in three different N-P-K treatments in the Keitt plots, Florence, South Carolina, September 9, 1949, variety Coker 100W. Differences associated with different amounts of leaf covering around the bolls.

| Treatment (N-P-K) | Percentage of bolls | | | | | |
|---|---|---|---|---|---|---|
| | To opening stage | | | | | |
| | Degree of fungus discoloration of fiber | | | | Insect-damaged | Not to opening stage i.e. immature |
| | None to slight[a] | Moderate | Severe | Very severe | | |
| 4-8-4 | 76.5 | 6.0 | 0 | 1.0 | 6.5 | 10.0 |
| 6-8-4 | 55.8 | 11.4 | 4.8 | 11.0 | 8.3 | 8.7 |
| 10-8-4 | 24.0 | 6.0 | 9.7 | 32,4 | 7.4 | 20.5 |

[a] A rating of severity of tight lock was estimated for each boll on the basis of the percentage of the total lock surface showing fungus discoloration. Thus a boll with 0-25% of the lock surface discolored was placed in the "none to slight" class, one with approximately 25-50% discolored in the "moderate"class, one with 50-75% in the "severe" class, and one with 75-100% in the "very severe" class. The "very severe" class was made up in part of bolls which had rotted so completely as to entirely fail to open and thus includes all cases of the typical Diplodia boll rot. Insect-damaged bolls, were, in general, either much distorted in gross appearance or penetrated by conspicuous boll worm (8) entries.

It might be considered as a possibility that differences in leafiness in the different treatments influenced the degree of build-up of anthracnose inoculum on the plants during the growing season, as has been suggested by Weindling (29), and that this influence rather than the amount of leaf-cover around the bolls at the time of boll opening was the dominant factor in determining the amount of anthracnose tight lock in the different fertilizer treatments in the Keitt plots. While it seems logical to suspect that the more leafy plants did actually harbor more anthracnose infection during the growing season, it seems apparent also that the close proximity of widely different fertilizer treatments in these plots must have made possible a very considerable amount of anthracnose inoculum on the opening bolls of the small less leafy plants. This consideration, combined with the extremely low amount of tight lock in the 4-8-4 treatment, makes it apparent that while abundant inoculum may be a necessary requirement for the occurrence of anthracnose tight lock, leaf covering of the bolls at the time of boll opening is very probably an even more critical determining factor.

Chemical defoliation had a pronounced effect on the amount of fiber damage. Table 2 records results of ratings in undefoliated and calcium cyanamide-defoliated rows of cotton of the variety Coker 100W. The designation "pedicel rot" in the table refers to a special type of symptom

Table 2. Amount of fungus tight lock and boll rot in defoliated as compared with undefoliated cotton (Coker 100W) at Florence, South Carolina, 1949.

| Treatment and date of observation | Percentage of bolls: To opening stage: Fungus discoloration: None to slight[c] | Moderate | Severe | Very severe | Pedicel rot | Insect-damaged | Not to opening stage i.e. immature |
|---|---|---|---|---|---|---|---|
| Sept. 13[a] | | | | | | | |
| Defoliated | | | | | | | |
| Section 1 | 65.3 | .8 | 1.6 | 3.1 | 0 | 7.9 | 21.3 |
| " 2 | 67.0 | 7.8 | .9 | 10.4 | 0 | 3.5 | 10.4 |
| " 3 | 68.0 | .8 | 5.0 | 5.0 | .8 | 4.2 | 16.2 |
| " 4 | 61.3 | 9.3 | 0 | 4.0 | 0 | 8.0 | 17.4 |
| " 5 | 59.8 | 1.1 | 2.2 | 10.9 | 0 | 7.6 | 18.4 |
| AVERAGE | 64.3 | 4.0 | 1.9 | 6.7 | .2 | 6.2 | 16.7 |
| Undefoliated | | | | | | | |
| Section 1 | 28.5 | 2.3 | 2.3 | 17.7 | 14.6 | 6.1 | 28.5 |
| " 2 | 34.5 | 2.3 | 8.0 | 23.0 | 8.9 | 4.6 | 19.6 |
| " 3 | 38.0 | 2.5 | 6.3 | 35.4 | 6.3 | 5.1 | 6.4 |
| " 4 | 30.9 | 2.1 | 4.2 | 29.8 | 9.6 | 16.0 | 7.4 |
| " 5 | 27.4 | 1.2 | 2.4 | 21.4 | 25.0 | 20.2 | 2.4 |
| AVERAGE | 31.9 | 2.1 | 4.6 | 25.5 | 12.7 | 10.4 | 12.9 |
| Sept. 7[b] | | | | | | | |
| Defoliated | 50.4 | 2.9 | 0.8 | 5.3 | 0 | 4.1 | 36.5 |
| Undefoliated | 38.0 | 10.0 | 6.2 | 17.6 | 0 | 3.7 | 24.5 |

[a] Counts of all bolls in each of five 10-foot sections for each treatment.
[b] Counts of 400-450 bolls on random-selected plants for each treatment.
[c] For explanation of system of rating see footnote [a] of Table 1.

complex in which there occurred a disintegration and rotting of the pedicels, usually accompanied by failure of the bolls to open. Lyle (13) has also noticed a pedicel rot of cotton, associated in his case with Sclerotium rolfsii.

A second instance of reduction of the amount of tight lock and boll rot by chemical defoliation was seen on the Hill farm near the Pee Dee Experiment Station in an insecticide-defoliant experiment carried out by Mr. F. F. Bondy of the station staff. In this case the check area was on somewhat uneven ground and the plants differed considerably in leafiness and in degree of natural defoliation from one part of the area to another. Readings taken in different parts of the check area clearly reflected differences in the degree of leafiness of the plants. Thus in the portion of the check area identified in Table 3 as "IA", the plants showed a fairly considerable amount of natural defoliation, whereas in a small part of the same area identified as "IB" which was in a low part of the area, the plants were much more leafy. The amount of fiber damage in the two cases was clearly related to the amount of leaf covering around the bolls. Defoliated plants in sections II and III showed almost complete control of fungus tight lock (Table 3).

Attempts to observe varietal differences in susceptibility to the production of tight lock and boll rot resulted in the general conclusion that while such differences very probably do exist, environmentally-associated differences in the amount of leaf cover around the bolls are often sufficiently important to over-shadow or mask varietal differences. A group of varieties differing widely in fiber properties and in genetic make-up was grown at Florence in 1949 for certain

Table 3. Influence of defoliation on the amount of tight lock and boll rot in an insecticide-defoliation experiment on the Hill farm, Florence, South Carolina, September 8. Insecticide used, benzene hexachloride.

| Treatment | Percentage of bolls | | | | | | |
|---|---|---|---|---|---|---|---|
| | To opening stage | | | | | Not to opening stage i.e. immature | Total no. of bolls counted |
| | Fungus discoloration | | | | Insect-damaged | | |
| | None to slight[a] | Moderate | Severe | Very severe | | | |
| IA No insecticide, no defoliant | 10.7 | 4.7 | 6.0 | 19.1 | 42.1 | 17.4 | 252 |
| IB Same as IA, but low ground and more leafy plants | 3.1 | 1.9 | 8.2 | 42.1 | 33.3 | 11.4 | 159 |
| II Defoliated no insecticide | 49.3 | 1.5 | 0 | 1.5 | 32.5 | 15.2 | 203 |
| III Defoliated and insecticide | 61.0 | 1.9 | 0.5 | 0.5 | 17.1 | 19.0 | 210 |

[a] For explanation of system of rating, see footnote [a] of Table 1.

seed composition studies. The varieties were each grown in plots 3 rows wide and 60 feet long, with replicate plots for each variety in each of 4 separate ranges. Except as is otherwise noted in Table 4 the ratings were all made in the third range. The plants were quite uniform in height at the time of taking the ratings, mostly 36 to 44 inches tall. None of the varieties was extremely rank in growth. The tight lock in this material was almost exclusively associated with Diplodia.

The data of September 4-5 in Table 4 from the "seed composition test" cotton seemed at first to indicate fairly clearly that some varieties are less susceptible to tight lock than others. The Coker 100 strains and Empire WR, for example, were unusually low in tight lock and boll rot by comparison with the other varieties. The degree to which this superiority may be influenced by an environmentally-associated difference in leafiness, however, was seen on a later date, September 9, when ratings were made on Coker 100W 47-70 in both the second and third ranges, the plants of this variety in the former range being for some reason more leafy than in the latter and distinctly higher in amount of tight lock and boll rot. These data are also presented in Table 4.

A second instance which appears to demonstrate the importance of the amount of leaf covering around the bolls as compared with other factors was noted in comparisons between the amount of damage in three varieties in the "seed composition test" field and defoliated plants from the same seed, planted at the same time in a nearby field as a part of a variety test. Data taken on September 5 in this latter field, Table 4, indicated much less tight lock and boll rot than on plants of the same varieties in the "seed composition test" cotton. The variety Pandora, a variety with large leaves, was particularly notable for the decrease in amount of tight lock which it showed in the defoliated plots of the variety test as compared with undefoliated plants in the seed composition test, although both the other varieties rated also exhibited conspicuous differences. In a small area of the Pandora planting in the third range which had undergone defoliation as a result of attack by the Fusarium wilt organism, it was noted that the amount of tight lock was distinctly lower than in the rest of the plot. A rating on 119 bolls on consecutive plants in such a wilt-area yielded percentages of 62.2, 7.6, 5.9, 9.2, 5.9, and 9.2 (September 9, figures arranged in same sequence as in tables).

In the "seed-composition test" cotton it was observed that plants on the ends of the rows were commonly more leafy than plants more toward the middles of the rows and that correlated with this situation there was a distinct tendency for heavily sporulating Diplodia-infected bolls to occur on these end-of-the-row plants, whereas the less leafy plants frequently had dried-out Diplodia-infected bolls with many pycnidia but with no spores being discharged. Leaf covering appears

Table 4. Amount of fungus tight lock and boll rot in "seed composition test" cotton and in certain varieties in Pee Dee variety test, Florence, South Carolina. Ratings included all bolls on random-selected plants of each variety to a total of 200-250 bolls.

| Variety and date of rating | Percentage of bolls | | | | | |
|---|---|---|---|---|---|---|
| | To opening stage | | | | | Not to opening stage i.e. immature |
| | Degree of fungus discoloration of fiber | | | | Insect-damaged | |
| | None to slight[a] | Moderate | Severe | Very severe | | |
| | "Seed composition test cotton" (undefoliated) | | | | | |
| September 4-5 | | | | | | |
| Acala 1517W | 25.5 | 18.8 | 5.7 | 19.3 | 4.2 | 26.5 |
| Acala 4-42 | 15.5 | 15.5 | 11.5 | 30.0 | 13.0 | 14.5 |
| Coker Wilds | 22.5 | 5.2 | 3.3 | 13.1 | 5.2 | 50.7 |
| Coker 100W | 46.7 | 5.2 | 3.3 | 7.1 | 4.3 | 33.3 |
| Coker 100W 47-2 | 43.5 | 9.7 | 6.0 | 6.5 | 2.8 | 31.5 |
| Coker 100W 47-70 | 56.0 | 15.7 | 5.8 | 12.6 | 3.1 | 6.7 |
| Coker 100W 1950 BFS | 39.1 | 9.9 | 3.3 | 18.5 | 9.9 | 19.2 |
| CSS 9 | 21.6 | 9.9 | 12.6 | 28.8 | 6.8 | 20.3 |
| Deltapine 15 | 40.9 | 9.1 | 13.6 | 20.4 | 5.0 | 10.9 |
| EH 809 | 25.3 | 13.6 | 5.0 | 16.7 | 7.2 | 32.1 |
| Empire WR | 51.1 | 6.3 | 6.8 | 10.8 | 6.8 | 18.1 |
| Hybrid 1282 | 51.1 | 7.5 | 5.2 | 17.5 | 3.7 | 14.9 |
| Mesilla Acala | 29.5 | 9.4 | 8.5 | 23.9 | 4.7 | 23.9 |
| Pandora | 27.5 | 13.3 | 11.9 | 22.9 | 4.6 | 19.7 |
| Sealand 542 | 12.5 | 7.9 | 6.0 | 26.8 | 6.0 | 40.7 |
| Stoneville 2B | 36.2 | 8.3 | 7.0 | 24.9 | 9.2 | 14.4 |
| White Gold W | 52.4 | 5.8 | 8.6 | 11.0 | 4.3 | 17.8 |
| September 9 | | | | | | |
| Coker 100W 47-70 3rd range, light leaf. | 53.0 | 10.0 | .6 | 9.5 | 6.2 | 20.7 |
| Coker 100W 47-70 2nd range, heavier leaf. | 44.1 | 5.7 | 10.0 | 27.8 | 8.4 | 4.0 |
| | Cotton in Pee Dee Station variety test (defoliated) | | | | | |
| September 5 | | | | | | |
| Pandora | 33.5 | 1.9 | .5 | 5.3 | 8.7 | 50.0 |
| Sealand 542 | 34.1 | 2.4 | .9 | 4.7 | 7.1 | 50.7 |
| Stoneville 2B | 35.6 | 3.0 | 1.5 | 4.0 | 7.4 | 48.5 |

[a] For explanation of system of rating see footnote [a] of Table 1.

therefore to be important not only for infection but also for sporulation and the initiation of secondary cycles of infection.

A type of fungus tight lock observed infrequently at Florence in 1949 but more commonly in the two preceding years might be designated as "basal tight lock". This condition involves fungus damage to the lint and compactness of the basal portion of the lock only and appears to occur most commonly in certain varieties, particularly the Acalas, in which, under Florence conditions, the bolls may not open widely enough, even in moderately favorable weather, for the basal portion of the lock to dry rapidly. In more humid weather, the whole lock, rather than merely the basal portion, is attacked.

Although it has not yet been adequately studied, fungus attack on the bolls following premature dehiscence as a result of very dry weather in July is reported to have occurred at Florence in 1949. Examination of a sample from a large field where this type of damage was said to have been prevalent disclosed numerous severely distorted bolls infected with Fusarium and Alternaria, and in a lesser number of cases with Diplodia, Nigrospora, and Rhinotrichum. Analysis of the situation was rendered difficult by the complicating factor of boll worm entries on many of the bolls.

## Observations on Samples from Other Locations

For comparison with material from Florence, samples of cotton affected with tight lock or related types of damage were obtained from the 1949 crop at each of four other locations. Observations on the cotton from these locations were limited, but of interest in certain respects. They were as follows:

Baton Rouge, Louisiana: A sample from Dr. D. C. Neal included 98 bolls with discolored locks and 108 additional separate locks. Of these, 79 of the bolls and 60 of the individual locks showed Colletotrichum setae on the fiber. Diplodia was found on the fiber in 39 of the bolls and on 36 of the separate locks.

Tifton, Georgia: A sample of 47 bolls was examined. Diplodia was found to be responsible for many of the tight locks, with the anthracnose fungus occurring only in small amounts on a few locks.

Auburn, Alabama: Many of the tight locks in the 30-boll sample examined showed dark mycelium on the fiber, at first unidentifiable, but producing in a majority of cases the characteristic pycnidia of Diplodia after short-term incubation on water agar.

Stoneville, Mississippi: When bolls exhibiting tight lock or related symptoms were requested from the U. S. Cotton Field Station, Stoneville, Mississippi, the material sent to Beltsville exhibited a condition not typical of tight lock as seen at Florence but probably related to it. The carpels were severely rotted at the base and characteristically fell apart or could be easily pulled apart from the pedicel and from each other. Separation of the inner from the outer carpel walls was also common and characteristic. The symptom complex as a whole might perhaps be appropriately described as a "shattered boll". When tissue from the bases of the carpels was incubated on water agar, each of the 23 samples tested developed an outgrowth of Fusarium sp., whereas Diplodia, Alternaria, Cladosporium, and Colletotrichum gossypii were each also found in several cases.

## Discussion

Any attempt to define the concept of fungus tight lock must necessarily include recognition of the fact that compact or poorly fluffed locks (not discolored) and relatively low fiber strength can and do occur, either separately or together, in the absence of microbiological damage to the fiber. Observation of the micro-organisms or of discoloration attributable to them on the fiber appears to be a necessary condition for diagnosis of fungus tight lock. If one considers a wide range of genetically different cottons, poor fluffing appears, from numerous observations made during several years of cotton breeding at Florence, to be strongly correlated with long fibers; whether this relationship is a direct causal one or primarily one of genetic linkage is not known. Fusarium wilt, or other diseases or conditions which result in poor secondary wall development in the fiber, may also cause poor fluffing. Low fiber strength and high x-ray angle have been shown by Berkley and others (7) to occur under environmental conditions in which the cotton plant during the period of fiber development has not suffered from severe water stress. Thus, weak fiber may occur in moist areas for a reason entirely unconnected with microbiological attack.

Infection directly upon the fiber rather than through boll tissue distinguishes anthracnose and Diplodia tight locks not only from true anthracnose and Diplodia boll rots but also from certain other boll rots. The Aspergillus niger and Rhizopus nigricans boll rots of cotton are described by both Shapovalov (23) and Ray (22) as rots which arise most frequently following entry of the boll by the common boll worm (8). The Alternaria rot described by Ray (22) as the most important cause of boll decay in Oklahoma is stated to be commonly a secondary invader following the bacterial blight organism.

In addition to the facts already presented, certain information from the work of other investigators is related to the problem of control of fungus tight lock. An attempt to summarize this information is made in the following paragraphs.

Alteration of cultural practices or choice of varieties to make boll opening occur during drier seasons has occurred to several individuals as a practical possibility in reference to control of boll rot and tight lock. Mr. J. H. Turner at the Tifton station in Georgia has been interested in this solution and has pointed out that at Tifton drier weather often occurs within a few weeks after the period at which cotton now commonly opens there (26). Weather records for the Pee Dee area in South Carolina obviously point to the same conclusion. Average precipitation for August, September, and October for the four stations Conway, Darlington, Florence, and Kingstree for the years 1920-49 were 5.52, 5.02, and 2.33 inches respectively.

More adequate use and interpretation of weather-forecasts is a subject of current interest in respect to several cotton problems and one which might easily come to have a practical bearing on the matter of controlling fungus tight lock and boll rot. In part, interest has undoubtedly sprung from the intensive application of weather forecasts in the Plant Disease Warning Service, a plant-disease forecasting service for a few plant diseases which was developed by and is coordinated through the USDA Division of Mycology and Plant Disease Survey. A relatively new development is the release to the public by the U. S. Weather Bureau, through the Superintendent of Documents, of the 30-day weather forecasts used as a partial basis for the predictions of the Plant Disease Warning Service, such weather forecasts now being issued on the 1st and 15th of each month. As a preliminary to the 5-day and shorter-term forecasts, the 30-day service, even though definitely less accurate than the shorter-term predictions, may have some value in predicting the probability of wet weather at the time of boll opening and the consequent urgency of preventive measures.

Inherent bacterial blight resistance as an aid in the control of boll rots is currently of interest to certain pathologists and may possibly have a bearing on the tight lock problem. It is to be pointed out at once, however, that in the case of tight lock, fungus attack occurs directly on the exposed fiber and that no preceding infection by bacteria would seem to be necessary for fiber attack by fungi. The numerous observations of bacterial invasion as a precursor of Diplodia (10) and Colletotrichum (31) infection on the outer surface of the bolls indicate that bacterial blight resistance might have some relation to the building up of Diplodia and anthracnose inoculum during the season. Such resistance has recently been incorporated into certain cotton breeding stocks by Simpson and Weindling (24) and more recently by others. Leafiness of the plants could also be important in this building up, as was suggested in the case of anthracnose by Weindling (29). Distribution of inoculum from a few sites of early-season infection in a field by insecticide dusting is also a possibility which probably can not be dismissed without evidence. The amount of over-wintered Diplodia spores in a given field in relation to winter temperatures and to the prevalence of infections in the same field in a previous season may also be important.

Incidence of boll rot and related fungus damage to cotton has been believed by several individuals to be influenced by defoliation, even though no quantitative estimates on the matter seem to have been published. A related concept which has also been put forth indicates that defoliation somehow forces bolls to mature and open at an earlier stage than would occur in the absence of defoliation. Martin and Leonard (16, p. 914), for example, reflect a common belief (2) when they state in respect to chemical defoliation that "The bolls ripen and open much more rapidly after the leaves drop." (Italics by the present authors). It may be of interest to note that a parallel belief in regard to hastening of boll maturation by defoliation was common some years ago in respect to defoliation of cotton by the Army worm and that when the idea was tested out in resulting experiments by Ludwig (12), manual defoliation materially lengthened the period required for boll maturation or left it unaffected. The data of Table 2 and of Table 4 (varieties Pandora, Stoneville 2B, and Sealand 542) seem to suggest an increase rather than a decrease in the time required for boll maturation as a result of defoliation. Further and more critical data should result from experiments on cotton defoliation now being carried out by other workers in several locations. It is the authors' present belief that when bolls have reached the dehiscence stage they are highly susceptible to primary fungus damage of the undried fiber and that in damp weather defoliation very frequently permits such mature bolls to open and fluff promptly rather than to rot or to become affected with fungus tight lock. They are not convinced that defoliation hastens maturity to the dehiscence stage.

## Abstract

Fungus attack resulting in major losses in both yield and quality of cotton fiber was observed at the time of boll opening in 1949 in the vicinity of Florence, South Carolina. The difficulty was caused in large part by Diplodia sp. and Colletotrichum gossypii, both of which were associated

with a general type of symptom complex here described and designated as "fungus tight lock". Typical Diplodia boll rot was also very common. The probable importance of anthracnose (C. gossypii) tight locks as a source of inoculum for seedling infection is noted.

Of the several factors which influence the incidence of fungus tight lock, the density of leaf cover around the bolls appears to be of predominant importance. Chemical defoliation of the plants resulted in major decreases in the percentage of bolls with Diplodia boll rot and with Diplodia and Colletotrichum tight lock. Originally suggested by workers at Florence (11), chemical defoliation has subsequently been believed by several individuals to "prevent boll rot". It has not been at all clear, however, just which ones of the several widely different disease complexes commonly referred to by this general term may be so controlled. The present paper attempts to provide at least a partial answer to this question. Supplementary observations on fungus tight locks from Alabama, Georgia, Mississippi, and Louisiana are also included.

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DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND SOUTH CAROLINA AGRICULTURAL EXPERIMENT STATION

# BOLL ROTS OF COTTON IN SOUTH CAROLINA IN 1949

C. H. Arndt

A survey was made in South Carolina during the week of September 11, 1949, to ascertain the prevalence and etiology of cotton boll rots as a result of several reports that indicated an unusually high incidence of such rots in several counties of the State. The observations in this note supplement those in the preceding paper by Marsh et al.[1], in that they indicate the relative distribution throughout the State of the boll rots and lint discoloration studied intensively by these workers at Florence, South Carolina.

The percentages of bolls that did not open normally and in which all or part of the seedcotton remained in the "tight-lock condition" as the direct result of infection of the bolls by fungi were estimated to be as follows: Aiken-Orangeburg County area, 25 to 90 percent; Florence-Lee-Sumter County area, 10 to 50 percent; and for the Piedmont, 1 to 20 percent. In the Piedmont the effect of boll rots on yields was generally negligible, but heavy infestation by the boll weevil reduced yields below that which might have been expected from the set of bolls. In this region most of the rotted bolls were infested by the boll weevil, while in the Coastal Plain there was no evident correlation between weevil injury and the incidence of boll rots. The prevalence of boll rots in the Coastal Plain and weevil injury in the Piedmont was reflected in the relatively low mean yield of 211 pounds of lint per acre in 1949 as compared to an average yield of 309 pounds per acre in the preceding 10 years. The lint grade was also correspondingly lower and the percentage that graded middling or better was only 50 percent as large as in 1948. At the time this is written data on yields and lint grades for the several areas of the State are not available.

For the State as a whole, the losses from boll rots in 1949 were probably greater than for any year since 1928. In one planting of 700 acres in Aiken County, practically all bolls were rotted and little seed cotton was obtained; although the set of bolls had been adequate for a yield of a bale per acre. In an adjacent planting of a variety that had less dense foliage, the percentage of rotted bolls was only about 1/3 as great.

The predominating pathogen was Colletotrichum gossypii. It was found on the inside of the carpels and lint of cracked bolls (Dr. Paul B. Marsh first called the attention of the author to the presence of C. gossypii on the lint), in lesions on unopened bolls, in lesions on the bracts and calyces at the bases of both rotted and unrotted bolls, and in lesions on small stems, petioles, leaves, and peduncles. C. gossypii was more prevalent on the bracts, calyces, and peduncles than in years of lesser rainfall, when Fusarium spp., Ascochyta gossypii, Cercospora gossypina, and an Alternaria sp. tend to be relatively more abundant on these parts of the plant.

Diplodia theobromae was present on many of the rotted bolls and lint of cracked bolls collected in Aiken, Florence, and Orangeburg Counties. It was rarely found on collections in the Piedmont. Fusarium spp., F. moniliforme (white mycelia), and a species forming salmon-colored mycelia (apparently F. roseum of Snyder and Hansen), formed the third most abundant group of fungi on the rotted bolls; and they were present on about the same percentage of bolls in all collections. In all fields in which the incidence of boll rots was as great as 20 percent, typical angular-leaf-spot (Xanthomonas malvacearum) lesions were present at least on the older leaves.

Field observations indicated that the cool, rainy weather that followed the period of unusually high temperatures from July 24 to 30, predisposed bolls to infection by the fungi associated with the boll rot. Reports indicate that many of the larger bolls developed cracks at the lines of dehiscence during the period of high temperature. This premature exposure of the interior of the bolls to direct infection by fungi was undoubtedly responsible for bolls in which the lint remained in the "tight lock" condition. There appear to be no similar previously published reports of boll "cracking" during periods of unusually high temperatures.

The high incidence of boll rots appeared also to be definitely associated with well distributed and, in part, abundant rainfall during July and August that resulted in development of larger plants than usual. Generally, the boll rots were more severe in the fields with the larger plants and on varieties with the heavier foliage.

SOUTH CAROLINA AGRICULTURAL EXPERIMENT STATION, CLEMSON

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# ROOT ROT DISEASE-COMPLEX CAUSING TOBACCO PLANT BED FAILURES IN PITTSYLVANIA COUNTY, VIRGINIA

Wilbert A. Jenkins

In common with a wide area of the fluecured belt, tobacco growers in Pittsylvania County are experiencing losses in their plant beds during the current season ranging from moderate to severe. In several instances, losses have been so high that failure of the beds resulted. In most instances reported, a good stand of plants was secured earlier, but continuing losses of young seedlings have brought many beds to the brink of depletion.

Of those beds seen, including approximately 1,000 square yards of bed area at the Tobacco Research Laboratory, Chatham, more or less extensive areas within the beds are either denuded of plants or are populated by stunted and dying seedlings. Aside from stunting, the most prominent foliage symptom shown by affected plants is etiolation and yellowing of the cotyledons. In time, if the plants survive for such a period, the true leaves become affected and show gradual development of a lemon- to orange-yellow color. Seedlings as large as a 50-cent piece have recently been found showing lemon-yellow color on two or more of the true leaves. Affected areas within plant beds increase in size with conditions not well understood at this time, resulting in the development of symptoms in and death of plants of comparatively large size. In several instances common weeds are being killed, particularly sweetclover (Melilotus sp., probably M. officinalis). On sweetclover, early symptoms are expressed by marginal etiolation resembling potash hunger symptoms, followed by complete etiolation and death of the plants. Tobacco seedlings, being so fragile at this season, disappear soon after death, leaving no trace of their presence after a day or so.

Since a vast majority of the affected beds examined (as well as those reported but not seen) had been treated with calcium cyanamide during last fall for pre-emergence weed control, it was first thought that injury was due to the direct action of this material or to fertilizer burn. More careful examination of the root systems, however, demonstrated the presence of typical root lesions, blind rootlets, and root necrosis, so frequently associated with nematode feeding. Root lesions varying in color from creamy-translucent to dark brown are present at intervals along the portions of the root system still present and are particularly noticeable near the root tips. In severely affected seedlings, very few roots to a complete absence of roots may be noted. In the roots remaining, particularly at the stubs of blind roots, discolored lesions are quite evident, accompanied by slight enlargement of the tissues of the lesion. This symptom is very striking on those seedlings which have survived long enough to develop a fascicle of roots from the hypocotyl. Most fatalities have been noted in seedlings showing lesions of the hypocotyl fascicle.

On teasing fresh lesions apart, enormous numbers of nematodes have consistently been found infesting them. In the hypocotyl area, up to 12 individual nematodes have been dissected from a single lesion.

On finding the nematode association with tobacco and sweet clover seedlings showing the symptoms described above, larvae of the root knot nematode (Meloidogyne sp.) were suspected to be present along with certain forms unfamiliar to the writer. Specimens of tobacco and sweetclover seedlings were therefore submitted to Dr. Steiner of the Division of Nematology, U. S. Department of Agriculture, for determination. For the tobacco seedlings Dr. Steiner has reported the finding of pure cultures and enormous numbers per lesion of two undescribed nematode species belonging to the genus Panagrolaimus to the exclusion of Meloidogyne species, and stated his opinion that these forms may be true pathogens capable of causing the symptoms under discussion. On the sweet clover roots one of these Panagrolaimus species was also present but in smaller numbers while a Chiloplacus species was extremely numerous. This genus is related to Acrobeloides which has been found associated with root lesions and root necrosis of forest nursery seedlings.

The writer entertains serious doubts that the disease described above is a new disease. The symptoms and ultimate course of the disease so closely approximates those which have been attributed to minor element deficiencies or toxicities that it could have been confused with these in times past. Neither is outright criticism being made of the use of calcium cyanamide on tobacco plant beds, in spite of the fact that previous work has shown that soil temperatures below 70° F., together with relatively high calcium, are very definitely correlated with population increase of members of the meadow nematode group infesting the roots of tobacco. It is entirely possible that exaggerated effects of root mutilation were promoted by the excessively dry weather experienced over a wide area during the current plant bed season.

This occurrence is being called to the attention of tobacco pathologists in the hope that partial to serious plant bed failures may be investigated elsewhere during the current season for the

presence of the symptoms described and for the possible association of root-infesting nematodes with such symptoms.

VIRGINIA AGRICULTURAL EXPERIMENT STATION, CHATHAM, VIRGINIA

## CANAIGRE LEAF SPOT

J. G. Brown and Alice M. Boyle

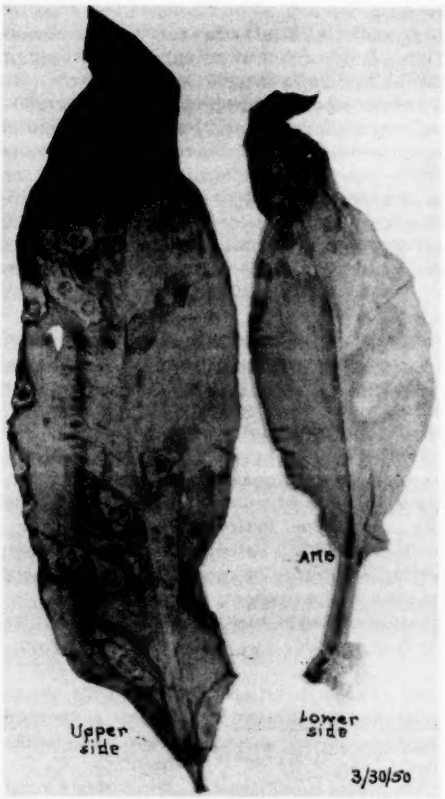


Figure 1. Leaf spot of canaigre, Rumex hymenosepalus Torrey, caused by Ovularia canaegricola Paul Henning.

Canaigre, Rumex hymenosepalus Torrey, is described by Bailey (1) as being distributed from Oklahoma to California. The plant is common in washes and the higher mesas in Arizona where it is often among the first herbaceous plants to become green in early spring. Its roots are sometimes fed upon by the wild peccary, -- an observation made by the senior author while dressing one of the animals some years ago. On account of the tannin content of the root, canaigre has been occasionally planted in Arizona and elsewhere. Cultivation of the plant and its subsequent disease explains the publication of this note.

Some weeks ago there were brought to us from an experimental plot specimens of canaigre showing diseased leaves. The leaves had spots somewhat resembling Alternaria spots on susceptible, broad-leaved plants. However, cultures of the affected areas, on potato-dextrose agar, gave very numerous sporulating tufts of Ovularia canaegricola Paul Henning.

Symptoms of Ovularia leaf spot on our specimens agreed with Henning's description (2), likewise the cause. Spots on green leaves of canaigre are pale yellow to light brown with somewhat lighter center, translucent, up to 2 cm. in diameter, with greenish yellow margin. By coalescence they become larger. The causal fungus has tufted, erect, simple (mostly) to bunched and curved conidiophores up to 80 $\mu$ in length. Conidiospores are elongated oval to club-shaped, less frequently ellipsoid, 15 to 24 $\mu$ by 8 to 11 $\mu$, hyaline, internally granular. We add to Henning's description that, on dry leaves (Fig. 1), the lighter centers of the spots are often in less contrast to the surrounding area, or even darker than the latter, as compared with the centers of spots on green leaves.

Ovularia leaf spot of canaigre and its cause were first described on leaves of Rumex hymenosepalus growing in the Berlin Botanical Garden, in 1897, and the cause was named Ovularia obliqua var. canaegricola. In 1897 the present name was published.

Since the leaf-spot fungus of canaigre is spread by aerial conidiospores, spraying the host plant with Bordeaux mixture or other good fungicide should protect against Ovularia leaf spot.

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UNIVERSITY OF ARIZONA, TUCSON

# SOFT ROT IN PEPPERS AND ITS TRANSMISSION BY THE CORN BORER[1]

C. D. McKeen[2]

Figure 1. Pepper plant with sufficient foliage removed to show healthy and diseased fruits.

Figure 2. Infection of pedicels with soft rot.

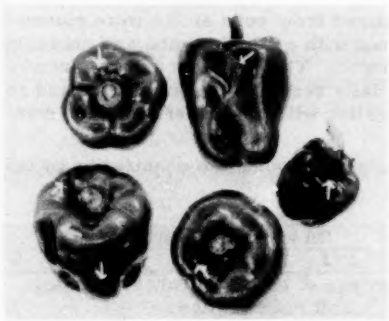

Figure 3. Pepper fruits manifesting soft rot infections. The arrows show location of larval entrances.

Soft rot caused by Erwinia carotovora Jones was reported by the writer (1) to be a serious disease of peppers in southern Ontario in 1947. Observations made during that season disclosed that soft rot became established in wounds caused by corn borer larvae and that the bacterial pathogen was largely dependent upon the insect for providing a suitable infection court. A recurrence of the disease in 1949 afforded the writer an opportunity to obtain additional information with respect to the disease-insect relationship. Certain recent observations and experimental results are reported here.

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[2]Associate Plant Pathologist.

## Occurrence and Symptoms

In 1948, soft rot in peppers occurred only to a limited extent in southwestern Ontario. In 1949, however, during late August, September, and October, the disease was widespread in most pepper fields and caused heavy losses. In two heavily-infected fields, 80 percent of the fruits that were set after mid-August were attacked and destroyed. Affected plants, such as shown in Figure 1, were commonly observed.

Most of the attacked fruits rotted in the fields while still attached to the plants. However, an appreciable percentage of fruits manifesting either no disease symptoms or scarcely recognizable insect injuries, when picked, decayed subsequently in transit to, or after arrival at, markets. The latter development of rot was sufficiently prevalent that markets became uncertain.

Although all peppers of the sweet and hot varieties commonly grown in southern Ontario have been attacked by soft rot, the disease has been more prevalent in sweet peppers of the bell and tomato types. Both green and ripe fruits are affected. Because a large proportion of the crop is harvested at the mature green stage, the disease appears to be more prevalent on green than on ripe peppers.

As mentioned in the earlier publication (1), soft rot of pepper fruits originated, with few exceptions, around the point of entrance of a corn borer larva. Typical examples of insect injury and soft rot infections are shown in Figure 3. The insect entered the pepper most frequently either by direct penetration of the fruit wall adjacent to the pedicel or through the pedicel. Less frequently, entrances were made elsewhere in the wall of the fruit. Small tunnels located in slight depressions in the fruit wall under the protecting portion of the pedicel were often so inconspicuous that they were not detected until incipient rot developed. Decay often extended from an affected fruit for a considerable distance down the pedicel (Figure 2).

## Experimental

To explain the occurrence of soft rot in 1947, it was postulated in the earlier publication that the insect was probably functioning as a vector in addition to providing the pathogen with a suitable infection court. To test the validity of this postulation, the following experiment was conducted.

After being examined carefully to ensure freedom from both soft rot and insect injury, green, sweet pepper fruits were surface-sterilized by immersion in an 85 percent solution of ethyl alcohol for 15 seconds and then dried immediately. Subsequently, the fruits were placed in enclosed metal containers with provision made for aeration. Corn borer larvae procured from pepper fruits manifesting either no rot or incipient soft rot infections were placed beside the peppers in one container. Larvae, of a similar number and size, obtained from corn stalks were placed in another tray. Other pepper fruits were aseptically punctured with a sterile probe and subsequently 50 percent of them were inocultaed with the soft rot pathogen. The containers were placed on the laboratory table for the duration of the experiment. A daily record of insect entries and soft rot development was kept. The details and results in connection with this experiment are summarized in Table 1.

Table 1. The development of soft rot in pepper fruits and its relationship to penetration by corn borer larvae.

| PEPPER FRUITS | | DAYS AFTERWARDS | | | | |
|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 |
| No. | Treatment | Percentage of larval entrances showing soft rot infections | | | | |
| 25 | Non-injured, 30 larvae from pepper fruits placed in containers | 40 | 66 | 81 | 84 | 84 |
| 25 | Non-injured, 30 larvae from corn stalks placed in containers | 6 | 16 | 19 | 34 | 35 |
| | | Percentage of injuries showing soft rot infections | | | | |
| 25 | Injured, inoculated with Erwinia carotovora | 72 | 92 | 100 | 100 | 100 |
| 25 | Injured, non-inoculated | - | - | - | - | - |
| 25 | Non-injured, non-inoculated | - | 4[a] | 4 | 4 | 4 |

[a] A small larva was found inside the only infected fruit in this treatment.

The results presented in Table 1 show that within a period of 5 days soft rot developed around 85 percent of the entrances made by larvae obtained from pepper fruits. It is also interesting to note that during a similar period soft rot developed in 35 percent of the entrances made by borers obtained from corn. Similar soft rot infections resulted when E. carotovora was introduced through probe wounds. In most infections, soft rot developed rapidly and complete collapse of the fruit occurred within 48 to 72 hours after infection was initiated.

Inasmuch as the results of the previous experiment have shown that corn borer larvae frequently were contaminated with soft rot bacteria and served as agents of dissemination and inoculation, and since the disease has been found to be almost entirely dependent upon the activity of the corn borer, it is apparent that disease prevention can only be attained by insect control. Thus, to safeguard the pepper industry in southern Ontario, the control of the corn borer in the pepper fields is essential.

Reference

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## DATA ON THE CONTROL OF GRAY LEAF SPOT OF TOMATO

Robert A. Conover

Tomato fungicide tests at the Sub-Tropical Experiment Station are primarily concerned with late blight (Phytophthora infestans DBy.) control. However, during the winter of 1948-1949, gray leaf spot (Stemphylium solani Weber) became epidemic in the plots and thus afforded an opportunity to obtain information on the effectiveness of certain fungicides for the control of this disease. Gray leaf spot was the only important disease present, late blight and early blight appearing only in trace amounts in the checks. The data obtained may be of interest in view of reports that gray leaf spot caused some damage to tomatoes in the middle-west during the summer of 1949.

The plots were set out on November 17, 1948, with Grothen Globe tomatoes, each plot being approximately one-hundredth of an acre in area and containing 36 plants. Each treatment was replicated four times in a randomized block design. Fungicides were applied every seven days (total of 11 applications) with a tractor-drawn power sprayer equipped with drop booms. Three nozzles per row were used while the plants were small, the number being gradually increased to nine with increase in plant size. Toxaphene was used as needed to control insects and was applied as a separate blanket spray to all plots. Mature-green tomatoes were picked eight times at weekly intervals beginning January 10, 1949.

The data obtained, presented in Table 1, strongly confirm the results of Dr. J. M. Walter published in the MARKET GROWERS JOURNAL, November, 1948.

Table 1. Incidence of gray leaf spot and yields of mature-green tomatoes from the 1948-1949 fungicide test.

| Material and amount used per 100 gallons | Gray leaf spot count[a] | Yield Bu. per Acre | |
|---|---|---|---|
| | | Marketable | Increase over check |
| 1. Dithane Z78 (2 lbs) | 2.6 | 329.8 | 149.0 |
| 2. Dithane D14 (2 qts), $ZnSO_4$ (1 lb), lime (1/2 lb) | 1.5 | 327.4 | 146.6 |
| 3. Parzate Liquid (2 qts), $ZnSO_4$ (1 lb), lime (1/2 lb) | 2.0 | 326.5 | 145.7 |
| 4. No. 2 alternated with No. 11 | 13.2 | 310.7 | 129.9 |
| 5. CR 305 (2 lbs) | 29.2 | 309.5 | 128.7 |
| 6. Dithane D14 (2 qts), $ZnSO_4$ (1 lb) | 1.7 | 304.4 | 123.6 |
| 7. Magnesium ethylenebisdithiocarbamate (2 qts), $ZnSO_4$ (1 lb) | 2.2 | 302.0 | 121.2 |
| 8. No. 9 first 8 applications, then 3 applications of No. 2 | 19.2 | 281.7 | 100.9 |
| 9. Copper Compound A (4 lb) | 39.0 | 274.2 | 93.4 |
| 10. Copotox, (4 lbs) | 53.9 | 269.6 | 88.8 |
| 11. Phygon XL (3/4 lb) | 28.9 | 266.2 | 85.4 |
| 12. Parzate (2 lbs) | 20.8 | 265.0 | 84.2 |
| 13. Check | 68.3 | 180.8 | --- |
| Least significant difference at odds of 19:1 | 9.3 | 58.8 | |
| " " " " " " 99:1 | 12.5 | 70.7 | |

[a]Average number of spots per leaflet (6 leaflets per treatment counted)

It is clear from these data that all fungicides providing zinc ethylene bisdithiocarbamate, with the exception of Parzate, were outstanding in gray leaf spot control. It should be mentioned that observations made in this and other tests with this lot of Parzate indicated it to be sub-standard. Perhaps this is the reason that the use of this material resulted in a yield reduction out of proportion to the amount of gray leaf spot present. Phygon, Copper Compound A, Copotox, Parzate, and CR 305 did not result in commercial control of gray leaf spot in this test.

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# POWDERY MILDEW OF SNAPDRAGON ON THE PACIFIC COAST

Kenneth F. Baker and Neil A. MacLean

Powdery mildew (Oidium sp.) of snapdragon (Antirrhinum majus L.), hitherto unreported from the western United States, has been found producing economic damage to greenhouse-grown snapdragons in two Pacific Coast states. The disease was noted at Pullman, Washington, in the fall of 1948. In December 1949 it was found in three greenhouses of the Redwood City area of San Mateo County, California.[1]

The symptoms of the disease were typically those of a powdery mildew, with both leaf surfaces, but predominantly the upper, showing characteristic powdery growth. Some of the older leaves were wilted and necrotic as a result of infection. Stem infections were common on some varieties but not found on others; in many cases they were nodal, representing extension by growth of the fungus from leaves and petioles. No perithecia were found.

The disease may readily be distinguished from downy mildew (Peronospora antirrhini Schroet.) that is prevalent in California. Downy mildew is most common on seedlings, where it causes a characteristic paling and downward marginal curvature of affected leaves, and frequently the death of all aerial parts. On older plants the terminal shoot growth is checked and leaves become pallid. The fungus sporulates on the lower surfaces of leaves as a white, sparse to matted, downy growth. Powdery mildew produces its powdery, white, surface growth on upper and lower leaf surfaces and on stems.

All varieties in the Redwood City area were infected, some more severely than others. Our observations and grower reports indicate that Peace and Afterglow were most severely injured, although they developed few stem infections. Dorcas Jane, Ball's Hybrid White and Ball's Hybrid Yellow all showed severe stem and leaf infection. White (17) observed no differences in varietal susceptibility.

Preliminary observations show that this mildew can be controlled with sulfur dust under California greenhouse conditions. In eastern greenhouses (4, 5) a wettable sulfur spray (1 to 1 1/2 lb. per 100 gal.), plus a suitable spreader to insure complete coverage, is recommended. This is applied at 7 to 14 day intervals, covering both upper and lower leaf surfaces, from the time seedlings are pricked off until they are 12 to 16 inches high. Fermate and Parzate are relatively ineffective.

## History and Distribution

Powdery mildew has been reported on the native Antirrhinum orontium L. in Europe for many years. Castagne (2) apparently described Erysiphe orontii Cast. on it in southern France in 1851. Léveillé (7) described E. linkii Lév. on this Antirrhinum near Marseille, France and on the island of Corsica in 1851, and regarded E. orontii Cast. as a synonym. Salmon (15) treated both of these as synonyms of E. cichoracearum. Blumer (1) regarded E. linkii as a synonym of E. artemisae (Wallr.) Grev., but pointed out the resemblance of both to E. cichoracearum. Magnus (8) reported E. communis (Wallr.) Fr. on the same plant in the Austrian Tirol in 1897. This was considered by Salmon (15) as E. polygoni, apparently after examination of a specimen. Blumer (1) reported Oidium sp. on A. orontium in Switzerland, and stated that he had not seen any mildew perithecia on Antirrhinum. Nicolas and Aggery (12) reported an Oidium sp. as common on shoots of A. orontium infected by Peronospora antirrhini Schroet. at Toulouse, France in 1939. Perithecia of E. linkii were mentioned and presumably present on the plants. Passerini (14) reported Sphaerotheca castagnei Lév. on A. orontium in Italy in 1881. This fungus was not illustrated, but he referred to Plate VI, fig. 1 of Léveillé (7) which is of the Erysiphe cichoracearum type. The identity of his fungus remains in doubt, but it should be noted that specialists have referred some exsiccati of this species to other binomials, including E. cichoracearum.

Powdery mildew was reported as Oidium erysiphoides Fr. on Antirrhinum majus by Oudemans (13), but it has been impossible to verify this in any of the references cited. It apparently was first clearly reported on this host in England in March and April 1928, when it was found by W. Buddin in Buckinghamshire and by A. Beaumont at Newton Abbot, Devonshire (11). It was again observed in Cambridge in 1934, in Shropshire and Glamorganshire in 1937, and has also been observed in Hertsfordshire (10, 11). The Oidium sp. occurred on plants outdoors and under glass. A specimen on A. majus (Herbarium of the Pathology Laboratory, Ministry of Agriculture, No. 21; examined through the courtesy of W. C. Moore) collected in Hertsfordshire in 1939 was found

[1] First recognized by R. H. Sciaroni, Farm Advisor, San Mateo County.

to have conidiophores of the E. cichoracearum type. An Oidium sp. was reported on A. majus in the Neuchâtel canton, Switzerland in 1936 (9).

Oidium sp. was first reported in North America by Guba (6) in October 1936 on greenhouse snapdragons in Lexington, Massachusetts where the grower reported it had frequently occurred. It continues to be a problem in that State (17). Powdery mildew was reported by Dimock (4) in New York greenhouses in 1941, but was present even earlier. It killed much of the lower foliage of the plants and sometimes spread upward into the flower spikes. The disease has continued to be destructive in that State (5) and has also been reported from Pennsylvania (16). The Oidium is present in the Midwest and caused considerable damage in a West Chicago, Illinois greenhouse range during 1949-50.

The powdery mildew apparently first reached the Pacific Coast in 1947, when it was found by W. Jones in a greenhouse at Sooke, British Columbia, causing slight damage (3). This was the first Canadian report of Oidium sp. on this plant.

Identification of the fungus in the United States must be provisional and based on the conidial stage. Fortunately this stage of E. cichoracearum and E. polygoni differ considerably (1; fig. 2A, 1 and 3). Examination of specimens collected in New York[2, 5], Illinois[3, 5], and in California[4, 5] showed the conidiophores to be of the Erysiphe cichoracearum type.

## Discussion and Summary

Powdery mildew on Antirrhinum has been known in Europe since at least 1851, and definitely on snapdragon since 1928. It was not, however, reported from North America until 1936, or the Pacific Coast until 1947. Since most of the early snapdragon seed was produced in Europe, this delayed spread indicates that seed transmission did not occur. Further evidence is afforded by the fact that no perithecial stage has been found on A. majus, and that conidia of Oidium sp. are notoriously short-lived. The evidence afforded by the rate of spread also suggests that the Oidium on snapdragon must be quite specialized, or it would have spread to this crop from other plants. It seems probable from these considerations that powdery mildew was spread by shipments of snapdragon plants. Florists' snapdragons used to be vegetatively propagated and shipped as cuttings. It is now common practice to plant in glasshouse benches snapdragon seedlings produced by a specialist. Such plants commonly are shipped into California from midwestern or eastern States.

There is some uncertainty concerning the identity of the powdery mildews on Antirrhinum. It seems clear, however, that Erysiphe cichoracearum DC. has been reported on A. orontium in Europe under the names E. linkii, E. orontii, and perhaps also as E. artemisiae and Sphaerotheca castagnei, and that E. polygoni DC. has been reported as E. communis. No perithecial stage has been reported on A. majus, and none on any Antirrhinum in North America. The type of conidiophore found on specimens from various parts of the United States and from England indicates that an Oidium sp. of the Erysiphe cichoracearum type is the common mildew.

It will be of interest to observe whether snapdragon powdery mildew will spread to outdoor commercial and garden plantings in California, particularly to the extensive seed fields of the State.

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[2]Collections on A. majus by A. W. Dimock at Corfu, N. Y. on Nov. 10, 1942, and in the Cornell greenhouses, Ithaca, N. Y. in March, 1948.
[3]Collected by V. Ball, West Chicago, Illinois, March 15, 1950.
[4]Collection by R. H. Sciaroni, Redwood City, Calif., Feb. 8 and 18, 1950.
[5]Specimen in Herbarium, Division of Plant Pathology, University of California, Los Angeles.

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# FUNGI ASSOCIATED WITH CAMELLIA FLOWERS

Alice J. Watson

During the camellia flowering season just past, inspectors of the Bureau of Entomology and Plant Quarantine, supplementary to other duties, made a survey for the possible occurrence of the camellia flower blight disease in the States of Alabama, Florida, Georgia, Louisiana, Mississippi, North Carolina, and South Carolina. This disease, originally described from Japan, had heretofore been found in this country in California and Oregon, and from one garden near Atlanta, Georgia. The presence of the disease in the latter locality made a search for other infection areas in the South desirable.

In the course of the survey 150 collections were made of suspicious flower material and sent to the writer at Beltsville, Maryland, for examination. Standard culture techniques were followed, involving surface sterilization of petals with corrosive sublimate or Clorox and subsequent plating out on potato dextrose and water agar and moist chambers. The flower blight fungus, Sclerotinia camelliae Hara, was obtained once from a collection from Winnabow, North Carolina and once from Atlanta, Georgia, the locality whence the first eastern report of the disease had come. These isolations were compared with authentic cultures supplied through the courtesy of D. L. Gill of the Bureau of Plant Industry, Soils, and Agricultural Engineering, who has been studying the disease at the Bureau laboratory at Springhill, Alabama.

In the course of the search for the flower blight fungus on these 150 specimens, numerous other fungi appeared with great regularity in the cultures and were isolated for further study. Approximately 30 species were so obtained and a list of these follows with such identifications and comment as pressure of other duties has permitted. A considerable number of these forms are obviously saprophytic only, but some are just as definitely pathogenic in their behavior.

1. Alternaria tenuis Nees ex Cda. (Conidia brown 12-48 x 6-13 $\mu$)
2. Botrytis cinerea Pers. ex Fr. (Conidia 6-13 x 6-8 $\mu$).
3. Chaetomium spp. (Citron-shaped brown ascospores 4 x 3 $\mu$. Perithecia with straight as well as branched echinulate hairs. Another species with ascospores 3-9 x 3-6 $\mu$, with spiral and straight hairs on the perithecia)
4. Cladosporium herbarum (Pers.) Lk. (Conidia 6-15 x 3-4 $\mu$)
5. Colletotrichum gloeosporioides Penz. (Conidia 12-19 x 3-6 $\mu$; setae 96-128 $\mu$)
6. Curvularia sp. (Conidia 3-septate, brown, slightly curved, 22-26 x 11-13 $\mu$)
7. Cylindrosporium camelliae Gz. Frag. (Acervuli in black spots; conidia hyaline, straight with blunt ends, 13-19 x 2-3 $\mu$)
8. Diaporthe eres Nits (Ascospores hyaline, uniseptate, 8-12 x 3-4 $\mu$. Asci 38 x 12 $\mu$, approximately, in long-beaked perithecia in groups). (See Phomopsis)
9. Discosia artocreas (Tode) Fr. (Three-septate hyaline conidia 16-19 $\mu$, with cilia 9-12 $\mu$ near tips)
10. Epicoccum sp., probably E. nigrum Lk. (Brown globose echinulate conidia 16-27 $\mu$)
11. Fusarium poae (Pk.) Wr. (Conidia 3-septate, 28-41 x 3-5 $\mu$)
12. Gloeosporium sp. (Conidia 4-6 x 3 $\mu$ in light cream-colored drops)
13. Glomerella cingulata (Ston.) Spauld. & Schrenk (Ascospores 12-22 x 3-8 $\mu$; asci 54-64 x 10-11 $\mu$)
14. Laestadia sp., probably L. camelliae (Cke.) Berl. & Vogl. (Ascospores centrally swollen, 11-16 x 4-6 $\mu$; asci 54 - 12 $\mu$; perithecia 150 $\mu$)
15. Helminthosporium, sp., probably H. polyphragmium Syd. with shorter conidia (Conidia brown, 6-septate, typically 70-76 x 16-19 $\mu$)
16. Heteropatella sp., probably H. palustris (Brun.) Sacc. (Hyaline continuous conidia, 9-12 x 2-3 $\mu$, with appendages and borne in a membranous pycnidium)
17. Macrophoma sp. (Conidia 11 x 4 $\mu$)
18. Nigrospora oryzae (Berk. & Br.) Petch (Conidia brown ovoid, 16-23 x 14-20 $\mu$)
19. Papularia sphaerosperma (Pers.) v. Hoehn. (Conidia 9-12 x 7 $\mu$)
20. Penicillium spp. (Conidia globose, 2-3 $\mu$)
21. Pestalotia guepini Desm. (Conidia 4-septate, 20-27 x 5-6 $\mu$; 15-19 brown cells; setae 4-20 $\mu$)
22. Pestalotia funerea f. camelliae Brun. (Conidia 20-25 x 6-9 $\mu$; cilia 19-22 $\mu$)
23. Phoma camelliae Pass. (Conidia 4-7 x 3-5 $\mu$ in salmon tendrils)
24. Phoma tenuis Pass. (Conidia bacillar, 2.5-3.0 x 0.5 $\mu$)
25. Phomopsis sp. Imperfect stage of Diaporthe eres Nits. (Conidia: alpha 4-10 x 2-4 $\mu$, and beta 16-35 x 1 $\mu$)

26. Phyllosticta sp., probably P. camelliae West. (Conidia 9 x 5 $\mu$)
27. Phyllostictina sp. (Conidia with one appendage, 9-11 x 4-6 $\mu$)
28. Sclerotinia camelliae Hara (Spermatia in heads as in Penicillium but becoming brown. Spermatia globose to pear shaped, 3 $\mu$, with connecting isthmus attached. No sclerotia found)
29. Sclerotinia sclerotiorum (Lib.) DBy. (Conidia 13-18 x 6-8 $\mu$)
30. Sclerotiopsis concava (Desm.) Shear & Dodge (Conidia 6-8 x 1.5 $\mu$, hyaline, sickle-shaped). One of the imperfect stages of Pezizella lythri (Desm.) Shear
31. Sporonema sp. (Conidia continuous, hyaline, 5-11 x 1.5 $\mu$; conidiophores 12-16 x 0.8.1 $\mu$) One of the imperfect stages of Pezizella lythri (Desm.) Shear
32. Trichoderma sp., probably T. lignorum (Tode) Harz. (Conidia hyaline, globose, 3 $\mu$)

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# CAMELLIA FLOWER BLIGHT IN LOUISIANA

A. G. Plakidas

The blight was first found by C. M. Moss, nursery inspector for the State Department of Agriculture on February 26, 1950, on camellia flowers on exhibit at the Shreveport Flower Show. The disease was confined to the flowers of only one exhibitor, a nurseryman from Shreveport. Specimens of the affected flowers were brought by Moss to this department for diagnosis. The specimen consisted of about two dozen blossoms of white, pink and red varieties. The lesions were typical of the disease, as described by Hansen and Thomas[1]. A fungus fitting the description of Sclerotinia camelliae Hara was readily isolated from the petal lesions. Cultures were sent to Dr. D. L. Gill, U. S. Department of Agriculture Field Laboratory, Springhill, Alabama, and Dr. H. N. Hansen, University of California, and the fungus was positively identified as S. camelliae. Typical sclerotia developed on the affected flowers which were kept in a moist chamber outdoors for 21 days.

The exhibitor stated that these flowers were not grown in Louisiana, but were imported from California, obviously in violation of the State quarantine. All the camellia flowers at the Flower Show were seized and burned, and we felt happy over the fact that this potential source of infection was caught in time. But as we shall see, it was already too late.

On April 3, two separate specimens of one blossom each, were received for diagnosis. Both specimens were collected in the same private garden in Shreveport and were sent by the owner himself and by the inspector, Mr. C. M. Moss. Both were found on potted plants in a lath house, and Mr. Moss stated that he made a careful examination of the plants growing in the open without finding any sign of the disease. S. camelliae was isolated from both specimens and sclerotia developed on these blossoms which were kept in a moist chamber for two weeks. The owner of the garden stated that he had purchased container-grown plants from California over a period of several years, either directly or through the same Shreveport nurseryman on whose flower exhibit the disease was first found, and that he had observed symptoms similar to those on the above specimens on some of his camellia flowers in the same lath house last year. Thus it would appear rather certain that the blight has been in this particular garden for at least one year and probably longer.

Three additional specimens of fallen flowers bearing sclerotia typical of S. camelliae, were found since. One of these was found in the nursery of the man on whose flower exhibit the disease was first noted, and the other two in private gardens. Only few affected flowers were found. The first two of these three specimens consisted of one blossom each, and the third of three. Mr. Moss informed the writer that he has made a rather exhaustive inspection of commercial and private plantings in the Shreveport area, examining thousands of absisced flowers and found only the above with sclerotia.

Caddo Parish, in which Shreveport is located, has been placed under quarantine to prevent the spread of the disease to other parts of the State (assuming that it has not already spread), and a concerted effort will be made to eradicate it.

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[1] Hansen, H. N. and H. Earl Thomas. Flower blight of camellias. Phytopath. 30: 166-170. 1940.

# SUSCEPTIBILITY OF TALL AND MEADOW FESCUES TO HELMINTHOSPORIUM INFECTION

K. W. Kreitlow, Helen Sherwin, and C. L. Lefebvre

In recent years, considerable interest has developed in the use of tall fescue, Festuca elatior var. arundinacea (Schreb.) Wimm., for pastures and lawns. Although tall fescue is generally more resistant than meadow fescue, Festuca elatior L., to crown rust, Puccinia coronata Cda.[1], a misconception seems to have arisen that this species is generally more resistant to other diseases as well. This was recently emphasized when complaints of damage to tall fescue by Helminthosporium spp. were received from some areas in the South[2]. In order to clarify the situation, data collected independently at Beltsville, Maryland, and State College, Pennsylvania, are here presented.

Helminthosporium dictyoides Drechs., the net blotch fungus, is one of the most common pathogens of both tall and meadow fescues. Foliar attacks by this fungus range from small, scattered, oval, brown spots on the leaves to extensive, coalescing, net blotch-type lesions that may result in complete withering of leaves and ultimate death of the plant. Experiments conducted at Beltsville, Maryland, indicate that other species of Helminthosporium may also attack plants of tall and meadow fescues. The following varieties were tested for their reaction to the pathogen. In addition, plants of Lolium perenne L. and L. multiflorum Lam. were included.

| Festuca elatior var. arundinacea | F. elatior |
|---|---|
| Alta | Domestic commercial FC 23, 122 |
| K 31 | English commercial FC 23, 294 |
| | Minn. #1449 |

Seed of each variety was sown in four-inch pots of soil. When seedlings emerged they were thinned to ten per pot. Two pots of seedlings of each variety were inoculated with each of six cultures used in the test. A noninoculated control of each variety was included. In addition, older plants of Alta fescue growing singly in four-inch pots were inoculated.

In this experiment six cultures were used as test organisms. The cultures were identified as follows:

Culture #1 -- Helminthosporium sativum Pam. King & Bakke isolated from Festuca rubra L. collected at Arlington, Virginia. 1939
Culture #2 -- H. sativum from F. ovina sulcata Hack., Beltsville, Maryland. 1939
Culture #3 -- Another isolate from the above host.
Culture #4 -- H. dictyoides, from F. elatior var. arundinacea, Eatonton, Georgia. 1948
Culture #5 -- Helminthosporium spp., from Lolium multiflorum Lam., Tifton, Georgia. 1939
Culture #6 -- Curvularia spp., from F. rubra, Arlington, Virginia. 1939

Inoculum was prepared by growing each culture on carrot decoction containing 1 percent dextrose in 250 cc. Erlenmyer flasks. The fungus mats that developed were disintegrated in a Waring blendor and the suspension was sprayed on plants to be tested. Inoculated plants were incubated in a moist chamber until lesions started to form. They were then moved to a greenhouse bench for further observation.

From data presented in Table 1, it seems evident that Alta and K 31 fescues are not only susceptible to the isolate of Helminthosporium dictyoides obtained from Georgia, but in addition they are also susceptible to the three cultures of H. sativum used in this experiment. The Helminthosporium from Italian ryegrass was non-pathogenic to the species tested while the Curvularia culture produced only small inconspicuous lesions on leaves of Alta, K 31, and U. S. commercial fescues.

Tests conducted at the U. S. Regional Pasture Research Laboratory at State College, Pennsylvania involved the following strains of meadow and tall fescues:

[1]Kreitlow, K. W. and W. M. Myers. Resistance to crown rust in Festuca elatior and F. elatior var. arundinacea. Phytopath. 37: 59-63. 1947.
[2]Transmitted by John R. Hardison to Division of Forage Crops and Diseases. Beltsville, Maryland.

Table 1. Comparative susceptibility of fescues and ryegrasses inoculated with different cultures of Helminthosporium and Curvularia* at Beltsville, Maryland.

| Culture No. | Grasses tested: Fescue: Alta: Seedlings | Alta: Older plants | K 31 | U. S. commercial | English commercial | Minn. 1449 | Ryegrass: Italian | Perennial |
|---|---|---|---|---|---|---|---|---|
| 1 | 8 | 7 | 9 | 8 | 9 | 8 | 7 | 7 |
| 2 | 8 | 8 | 9 | 9 | 10 | 8 | 7 | 7 |
| 3 | 6 | 3 | 6 | 7 | 6 | 5 | 5 | 5 |
| 4 | 8 | 7 | 8 | 4 | 6 | 2 | 5 | 6 |
| 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 6 | 2 | 0 | 3 | 2 | 0 | 0 | 0 | 0 |
| Check | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

*0 = no infection, 10 = very susceptible

Festuca elatior var. arundinacea
N. Y. #2659, originally from England
Alta
K 31

F. elatior
S 53, Wales
Svalof early
Minn. #1449
Otofte, Denmark

Spores of H. dictyoides were obtained from susceptible plants growing in pots in a greenhouse. Infection was accomplished in two ways: (1) -- Spores collected from susceptible plants were atomized in aqueous suspension on leaves of plants to be tested. (2) -- Diseased and healthy leaves alternately were drawn between the moistened thumb and forefinger thus transferring spores from diseased to healthy plants.

Experience indicated that drawing leaves between thumb and forefinger was a slower, but more effective method of inoculation presumably because better wetting was effected. For the test, 18 to 20 older plants of each strain of fescue growing singly in four-inch pots were inoculated. The inoculated plants were incubated in a moist chamber for 48 hours, then placed on a greenhouse bench for observation.

Data presented in Table 2 demonstrate that none of the strains of tall fescue tested was resistant to H. dictyoides. These results agree substantially with the results of tests conducted independently at Beltsville, Maryland.

Table 2. Fescue strains tested and number of plants in each infection class at State College, Pennsylvania.

| Infection Class | F. elatior var. arundinacea: N. Y. #2659 | Alta | K 31 | F. elatior: S 53 | Svalof | Minn. #1449 | Otofte |
|---|---|---|---|---|---|---|---|
| Susceptible | 3 | 5 | 5 | 5 | 8 | 9 | 14 |
| Moderately susceptible | 6 | 7 | 5 | 11 | 8 | 10 | 6 |
| Slightly susceptible | 8 | 6 | 10 | 2 | 3 | 0 | 0 |
| Resistant | 2 | 1 | 0 | 0 | 0 | 0 | 0 |

Differences in reaction to H. dictyoides among plants of meadow and tall fescues in both field and greenhouse suggest the possibility of isolating plants resistant to the disease.

U. S. REGIONAL PASTURE RESEARCH LABORATORY, STATE COLLEGE, PENNSYLVANIA, AND DIVISION OF FORAGE CROPS AND DISEASES, PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND

# DISEASES OF FORAGE LEGUMES IN IOWA IN 1949[1]

J. M. Crall

RED CLOVER. Root rot or wilt of first year seedlings was a major factor in reducing stands of commercial plantings of red clover. Wilting associated with a vascular discoloration of the tap root was noted first in mid-May. The number of wilting plants observed at any time during the season was not large. However, vascular discoloration of 65 percent of the plants in one field occurred late in the season and vascular discoloration of 5 to 20 percent of field plantings was observed frequently after late June. In addition, a cortical rot of the tap root was observed in some fields after mid-August. The relationship between vascular discoloration and the cortical rot, if any, was not determined. Isolation attempts from both kinds of diseased tissue consistently yielded Fusarium oxysporum and frequently yielded other Fusarium species, including F. solani, F. moniliforme, and F. roseum. F. oxysporum also was isolated most frequently in April and early May from the discolored tissues of the crown and upper tap root of second-year plants but F. moniliforme was isolated most frequently later in the second season.

A number of the fungi isolated from the root and crown were tested for pathogenicity on the variety Emerson, seeded in infested soil in the greenhouse at 60° to 70° F. Ten of 22 isolates of Fusarium oxysporum reduced stands 35 to 62 percent after three months. The plants wilted and developed vascular discoloration typical of the Fusarium wilts. Several isolates of F. solani and F. moniliforme caused slight reductions in stand. An isolate of Phoma trifolii from the crown of a second-year plant caused 58 percent reduction in stand one month after planting. Although this organism was never isolated from the crown or root of first-year seedlings, surveys were not adequate to preclude the possibility of its presence there early in the season. It was found commonly in April on leaves and stems of second year plants.

The results of one year's survey and pathogenicity tests indicate that wilt and/or root rot caused by several Fusarium species, particularly F. oxysporum, may be a major factor in the reduction of red clover stands in Iowa, especially during the year of seeding. Phoma trifolii also may be important as a pathogen of young seedlings.

Three other diseases were important in 1949. The angular leaf spot caused by Cercospora zebrina was noted in early June. Maximum infection occurred during July and August, with a marked reduction in intensity of infection later in the season. The concentric-ringed leaf spot caused by Stemphylium sarcinaeforme was found in late June. It also occurred in maximum amount and intensity during July and August. These two leaf spots occurred in both first and second year plantings, but were most damaging in the former. Both occurred frequently in the same field. Leaf spot infection of 100 percent of the plants was not uncommon during July and August, and defoliation as a result of leaf spots was estimated as high as 50 percent in some fields. The common occurrence of Cercospora zebrina on red clover has particular significance since the organism was also very prevalent on alfalfa in 1949. Powdery mildew (Erysiphe polygoni) was also found in late June. Maximum infection occurred during August, and infection was quite prevalent, though in light intensities, in October. In some fields 100 percent of the plants were infected.

Diseases of minor importance in 1949 were: northern anthracnose (Kabatiella caulivora), southern anthracnose (Colletotrichum destructivum), spring black stem and leaf spot (Phoma trifolii), leaf spot (Stemphylium botryosum), and rust (Uromyces trifolii).

Of some interest was the unusual occurrence of a virus ringspot, reported originally from Kentucky in 1933,[2] but since unreported in this country. It was found at three locations. In one field 5 percent of the plants were infected.

ALFALFA. Downy mildew (Peronospora trifoliorum) was found commonly on both 1949 and older plantings. It was noted in slight amounts in early April. The amount and severity of infection were maximum about mid-May when 50 to 75 percent light to moderate infection was common and losses of the first hay crop were estimated as high as 30 percent. Downy mildew was not found commonly after mid-July, but plants in numerous fields showed a chlorosis suggestive of downy mildew infection throughout the season.

Common leaf spot (Pseudopeziza medicaginis) occurred from late May through the season. The amount and severity of infection varied markedly from field to field. In mid-August several fields with 100 percent severe infection, causing marked defoliation, were seen. In general, the disease was most damaging where plants were past the best cutting stage for hay or where plant vigor was lowered by other factors.

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[1]Journal Paper No. J-1773 of the Iowa Agricultural Experiment Station, Ames, Iowa. Project No. 1162.

[2]Johnson, E. M., A ringspot-like virus disease of red clover. Phytopath 23: 746-747. 1933.

Spring black stem (Ascochyta imperfecta) was found in small amounts in most fields in early April. It occurred in maximum amount and intensity, though still light, in early May, and was not found commonly after the first week in June.

Summer black stem and leaf spot (Cercospora zebrina) was noted in early July. Infection was abundant and severe in some fields during August and on old stems as late as the first week in October.

Diseases of minor importance were: anthracnose (Colletotrichum destructivum), bacterial wilt (Corynebacterium insidiosum), yellow leaf blotch (Pseudopeziza jonesii), and rust (Uromyces striatus medicaginis).

Root rots were not particularly damaging on alfalfa. Isolations from plants with root and crown discolorations yielded the following fungi: Fusarium oxysporum, F. solani, F. roseum, Pyrenochaeta sp., Fusarium moniliforme, and Rhizoctonia. None of these fungi was tested for pathogenicity on alfalfa.

MISCELLANEOUS LEGUMES. Rhizoctonia root and stem rot was a serious disease in experimental plantings at Ames on birdsfoot trefoil (Lotus corniculatus) and Ladino clover. It was more severe on the former. Most damage occurred during periods of hot dry weather. Rhizoctonia was also found infrequently during the season in commercial plantings of alfalfa, red clover, and soybean.

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## SEVERE COLD REDUCES RUST IN PINE PLANTATIONS

Willis W. Wagener

About 30 years ago several experimental plantations of ponderosa pine, Pinus ponderosa scopulorum, were made by the U. S. Forest Service in the South Fork drainage of Mink Creek, Caribou National Forest, southeast of Pocatello, Idaho. Losses of planted trees were fairly heavy during the early years of the plantations, but the surviving pines have made thrifty growth and have appeared to be well adapted to the site. Porcupines have been troublesome and would have caused very heavy damage had it not been for the protective action of local forest officers. In recent years, infections by a rust, Cronartium comandrae, have also appeared in damaging numbers, chiefly on the 1940 and 1941 growth of branches and on scattered main stems. When the plantations were visited in mid-July of 1948, rust cankers in full sporulation were very noticeable. Distal portions of many of the attacked branches were dead or dying, but the rust was still active as a rule in the remaining part of the respective branches.

The winter of 1948-49 was extremely cold in western United States and on low ground severe injury often resulted, even to hardy native conifers. In a few places, native ponderosa and Jeffrey pines up to 24 inches in trunk diameter were so badly injured that they died the following summer. At Pocatello Airport, Idaho, the closest regular Weather Bureau Station to the Mink Creek plots, the minimum recorded temperature for the winter was -31° F. on January 25, 1949.

When the writer revisited the Mink Creek plots on July 19, 1949 much dead foliage was immediately apparent, but a more detailed examination showed that practically all of it was on branches or main stems bearing rust cankers or girdled by porcupines. Branches bearing rust cankers were usually killed back to the main stem, and in almost every case the entire portion of the branch or stem invaded by the rust was dead. Living cankers were hard to find except in the upper part of the plantations which occupy a gentle easterly slope above the South Fork of Mink Creek. From the gradation in killing up the slope, it was evident that a temperature inversion had prevailed during the extremely cold period when the injury occurred and that most of the cankers at the upper levels had escaped complete killing because of the higher minimum temperatures there.

The net result of the cold spell has been the almost complete elimination of the comandra rust from all except the upper levels in the plantations. Practically all of the trees will survive the crown retrenchment accompanying the killing of cankered branches, and they are now of sufficient size to be almost free of danger of serious damage by the rust in the future. The severe cold has thus had a marked sanitary effect in the plantations.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

# THE DISTRIBUTION AND SIGNIFICANCE OF THE CHESTNUT ROOT ROT PHYTOPHTHORAS, P. CINNAMOMI AND P. CAMBIVORA[1]

Bowen S. Crandall[2]

The root disease of European chestnut, Castanea sativa Mill., is believed to have been present in Spain since 1726 (12) and was first reported in Portugal in 1838 and elsewhere in Europe soon after. In 1917 Petri isolated the causal organism and classified it as Blepharospora cambivora Petri (8), later changed to Phytophthora cambivora (Petri) Buis. Phytophthora cinnamomi Rands was first reported as associated with the disease in southern Europe in 1932 (5) and in England in 1936 (3). However, European pathologists have attributed the ink disease of chestnut to P. cambivora.

In the United States what is believed to have been the root disease of Castanea apparently was first observed in 1824 (2). In 1932 Milburn and Gravatt (7) reported a Phytophthora, tentatively thought to be P. cambivora, as the cause of the root disease of chestnut, C. dentata (Marsh.) Borkh. In 1945 Crandall, Gravatt, and Ryan (2) classified the causal parasite as P. cinnamomi.

In 1936 Mehrlich (6) suggested the combination of P. cambivora and P. cinnamomi into a single species P. cambivora. Crandall, Gravatt, and Ryan (2) opposed this combination as confusing and dangerous, since the host ranges of the two species did not, in general, overlap. The two species may readily be distinguished in culture, and it is only in the published descriptions and especially in subsequent publications on P. cambivora that overlapping characteristics are to be found. P. cambivora is recorded on a limited number of temperate zone hosts, chestnuts in Europe (8), beech in England (3), and maple in North America (10). P. cinnamomi is considered to be the species actually involved in Day's long list of hosts for P. cambivora (3), including walnut, rhododendron, American chestnut, among others. In part, this confusion resulted from White's attribution (16) of rhododendron root rot to P. cambivora, and from Crandall's listing (1) of a considerable number of coniferous and broadleaf hosts attacked by P. cambivora, with P. cinnamomi considered as a synonym. In both these cases the fungus considered was actually P. cinnamomi, now recognized to be a separate distinct species and recorded on a wide variety of tropical, subtropical, and temperate zone hosts.

The primary purpose of this note is to bring to the attention of pathologists some recent publications in Spain and Portugal which clarify the European situation considerably, by demonstrating that a considerable part of the chestnut root rot in Europe actually is due to P. cinnamomi and not to P. cambivora, and by enabling us to fix the host ranges of the two species with some certainty.

Urquijo Landaluze in Spain in 1942 (11) reported preliminary cultural studies in which he compared his Spanish Phytophthora isolates from chestnut and walnut with Phytophthora isolates received through the Centraalbureau voor Schimmelcultures, Baarn, Netherlands as P. cambivora. Included in the cultural studies from this source were isolates from C. sativa (from Petri), C. sativa (from Dufrenoy), C. dentata (origin not given), Erica sp. (origin not given), Rhododendron sp. (from Leonian) and Persea sp. (from Leonian). Reading his reports, one can easily visualize what happened. His own isolates were being compared with a group received under the name of P. cambivora, but which actually contained both P. cambivora and P. cinnamomi isolates. To solve the difficulty which would obviously confront him, he provisionally set up three groups. Additional studies were reported in 1943 (13) confirming not only differences between his Spanish isolates and those received as P. cambivora but differences among the P. cambivora isolates as well. In 1946 (14) resistance of the isolates to copper sulfate concentrations was reported. At this time P. cambivora was recognized and set apart and a residue group, including the Spanish isolates, tentatively thought to be P. verrucosa Alcock and Foister (4). Correspondence with the writer at this time indicated that, in spite of the confusion created by the names under which the cultures were received, Sr. Urquijo had already arrived at the conclusion, which the writer had surmised from the earlier publications, that he was dealing with a large group of P. cinnamomi isolates. In 1947 Urquijo (15) in a masterly summation, in which he reviewed his earlier work and the report on chestnut root rot in the United States (2), concluded that the chestnut root rot in Spain was caused by P. cinnamomi and not P. cambivora. The isolates of Petri and Dufrenoy only,

[1]A contribution from the Centro Nacional de Agronomía, a technical agricultural service organization for El Salvador, operated jointly by the Office of Foreign Agricultural Relations, U. S. Department of Agriculture, and by the Ministry of Agriculture of El Salvador. The funds supporting this work were provided by the U. S. Interdepartmental Committee on Scientific and Cultural Cooperation and by the Republic of El Salvador.

[2]Senior Pathologist, U. S. Department of Agriculture, and Chief, Department of Plant Pathology, Centro Nacional de Agronomía.

from C. sativa, remained to represent P. cambivora, while all other isolates including his local material were assigned to P. cinnamomi. He mentioned further that work carried out by Pimentel in Portugal had showed the presence there of the two species and that undoubtedly Petri and Dufrenoy and others have worked with both of them without realizing it. The presence of the two species in France and Italy might explain some of the overlapping characters reported from time to time for P. cambivora. Pimentel's work reported in 1945 (9) now assumes added significance. Earlier, he had set up a separate group (called Phytophthora A) to include strains isolated from root rot of chestnut, walnut, Douglas-fir, and cork oak, Quercus robur, in Portugese nurseries in 1941 and 1942. In 1943, this same fungus, which he was unable to relegate to P. cambivora, was isolated from cork oak (Quercus suber) and in 1944 from birch (Betula alba). Representatives of another group (called Phytophthora B) were isolated from chestnuts in a single nursery. Cross inoculations were made with the isolates on the primary and other hosts.

Pimentel concluded that his Phytophthora A was the same as Urquijo's residue group (i.e., not P. cambivora but another species and suggested more studies of P. cinnamomi with which it had considerable affinities) now positively identified as P. cinnamomi; while his Phytophthora B corresponded to Urquijo's first group, the authentic P. cambivora.

The results of the field isolations and inoculation studies take on considerable significance since they provide comparable trials of both species on a group of hosts, two native to the United States and four extensively introduced in the southern and West Coast States. The disease is not known or has not been reported in nature on Douglas-fir in the United States. The writer has viewed with some alarm the greenhouse demonstration of the high degree of susceptibility of Douglas-fir, Pseudotsuga taxifolia (Poir.) Britton, (2) to P. cinnamomi. This concern now seems justified by Pimentel's report (9) in 1945 of the natural occurrence of the disease in plantings in Portugal. In general, with this clarification of the status of P. cambivora and P. cinnamomi, a considerable fund of accumulated European information on host ranges, and by inference on resistant or immune hosts becomes available. This information should be especially useful to foresters and forest pathologists. Cork oak, for instance, has been extensively introduced into the United States within the known range of P. cinnamomi, both in the South and in California.

In addition to hosts reported from the United States and elsewhere and in earlier European publications, which certainly should now be critically reviewed, additions to the host range of P. cinnamomi can be established. From the publications herein reviewed, P. cinnamomi has been found to attack Castanea sativa, Juglans regia, Pseudotsuga taxilifia, Quercus robur, Q. suber, Betula alba, Cedrus atlantica, Abies nordmanniana, A. pectinata, A. siberica, and Castanea crenata var. tamba.

In contrast P. cambivora was found only on Castanea sativa and C. crenata var. tamba.

Thus we can assume that in Spain, Portugal, France, Italy, and elsewhere in southern Eurrope P. cinnamomi has played a much greater part in the chestnut root rot problem than was formerly realized; in fact, it probably is the major cause of the death of chestnuts in the more southerly regions. On other forest tree species the evidence points to it as being the pathogen. The rather considerable known host range of this fungus can thus be used as a criterion to govern the safety of planting or introducing tropical, sub-tropical, and temperate zone plants.

Within the United States we gain some additional assurance that P. cambivora will not attack birch or Douglas-fir, a danger that its possibly more northerly tolerance might have allowed. However, we have added evidence that the stands and nurseries of Douglas-fir must be closely watched, since Douglas-fir is susceptible to P. cinnamomi. It is located to a considerable extent within the climatic zones in which P. cinnamomi has been found naturally, and almost overlaps the range of P. cinnamomi on Persea americana, and the possibility is ever present that a strain of this fungus may encounter and attack Douglas-fir. Pimentel (9), and Crandall, Gravatt, and Ryan (2) pointed out that strain differences evidently exist but that the boundaries are poorly defined and can often only be recognized through slight differences in host susceptibility or resistance.

It would appear that P. cinnamomi is one of the world's major plant killers. A monographic study of this species is certainly indicated since its wide distribution makes it probable that a historical study of its movements and distribution on plant groups would aid considerably in the understanding of the modes of distribution of plant pathogens which, unlike many, do not possess spore forms easily carried by various means. It seems possible that P. cinnamomi is of Oriental, origin, perhaps reaching Europe during the time of the Crusades or even earlier, thence spreading through southern Europe to Spain and Portugal, and then throughout the New World during and following the age of conquest. Many inferences could probably be drawn by a study of its known hosts and their resistant or immune relatives.

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## BRIEF NOTES ON PLANT DISEASES

### ROSE RUST (PHRAGMIDIUM AMERICANUM?) IN LOUISIANA

By A. G. Plakidas

A specimen of severely rusted leaves of cultivated rose, variety "Fashion," was received recently from Newellton, Tensas Parish, Louisiana. As far as the writer knows, this is the first report of the occurrence of rose rust in the Deep South. No evidence was found in the files of this Department of a previous occurrence of rose rust in Louisiana. Of the 9 species of Phragmidium on roses listed by Arthur, only two range southward, P. speciosum as far south as North Carolina and P. americanum as far as Northern Mississippi. Newellton, Louisiana, lies in the latitude of Central Mississippi, about 20 miles south of Vicksburg. Only uredospores were found on the specimen.

DEPARTMENT OF BOTANY, BACTERIOLOGY AND PLANT PATHOLOGY, LOUISIANA STATE UNIVERSITY

### POWDERY MILDEW ON AFRICAN VIOLET IN PENNSYLVANIA

By R. S. Kirby

Powdery mildew (Oidium sp.) was found on African violets (Saintpaulia) on April 19, 1950, in Pittsburgh by L. P. Nichols. This is the first report of this fungus on African violets in this State.

PENNSYLVANIA STATE COLLEGE, STATE COLLEGE

### SOME DISEASES OBSERVED ON LEGUMES AND GRASSES IN KENTUCKY

By W. D. Valleau

Dying of Hairy Vetch. -- Hairy vetch is used extensively with a small grain as a cover crop following tobacco or in a continuous tobacco-vetch, small grain rotation. During the winter, which was unusually warm, and spring there were many reports of vetch dying. In all cases examined, the disease was black-stem presumably caused by a species of Ascochyta. This organism fruited on some of the stems in a damp chamber.

All of the vetch examined, except in thin stands or isolated plants, had stems that were very small in diameter and black for six or eight inches, but this was surmounted by a vigorous spring growth of larger diameter. In pure stands of vetch and winter weeds the blackening was superficial and did not kill the stems even though the blackened portion was in a "damp chamber" formed by the vetch and weed growth, nearly continuously. As late as May 8 what appeared to be vigorous shoots of vetch in heavy stands of barley were wilting and on examination the blackened bases of the stems were found decayed. In these instances shading by the barley and competition for nutrients seemed to have played a part as the bases of the stems were not in a more unfavorable condition than those in a solid stand of vetch.

Crown Rot of Crimson Clover. Crown rot (Sclerotinia trifoliorum) was severe on crimson clover over the State, probably because of a warm open winter with considerable rainfall.

Anthracnose on Orchard Grass. Two specimens of orchard grass were received from Northeastern Kentucky during the first week in May. In both instances, which seemed to be typical for the orchard grass plantings of the respective counties, the grass had started into growth in the spring, producing numerous leaves that were gradually killed by Colletotrichum cereale (C. graminicola). Some of the plants died at this stage. In others the buds which were to produce the culms were dead and lateral buds were pushing out small leaves that were quickly injured by anthracnose. In still others the culms were developing more or less normally but the basal leaves were all dead. The extent to which above-ground parts were developing indicated closely the extent of living roots. On plants from which there was no new growth developing the roots were all dead. Those pushing out a few weak leaves had an occasional live root while those growing more normally had several live roots amongst a mass of dead ones. The condition of the plants probably could not be attributed to anthracnose alone, although it was evidently the immediate cause of the death of the leaves. It seems probable the plants went into the winter in poor condition, as orchard grass is usually heavily infected with leaf spot organisms during the summer. The winter was very warm and one of the farmers said there had been some growth all winter, probably depleting the food reserves in the roots. Death of the leaves during the spring and depletion of food reserves may have been the immediate cause of death of the plants. One farmer reported similar death in the spring of 1949 following an open mild winter. The orchard grass was a strain that had been on the farm for about 50 years but evidently was not yet adapted

to a mild open winter. The reduction in stand varied from about 90 percent in some parts of the field to scattering plants in other parts.

On the Experiment Station farm all of the lower leaves had been killed, presumably by anthracnose, while the live foliage was confined to the culms. This was recognized by the agronomists as normal for this time of the year.

KENTUCKY AGRICULTURAL EXPERIMENT STATION, LEXINGTON

## SOME DISEASES ON SMALL GRAINS IN PENNSYLVANIA

By R. S. Kirby

Leaf rust (Puccinia rubigo-vera var. tritici) on winter wheat was starting to build up on April 28, 1950, in Franklin County. This is one of the earliest dates in the spring that leaf rust has been observed in this State.

Powdery mildew (Erysiphe graminis) is rapidly increasing on winter wheat this spring and may become destructive again.

Scald (Rhynchosporium secalis) was found on winter barley in York County on April 20, and in Lancaster County on April 28. Moderate to heavy infection was present on the barley plants, which were six to eight inches high.

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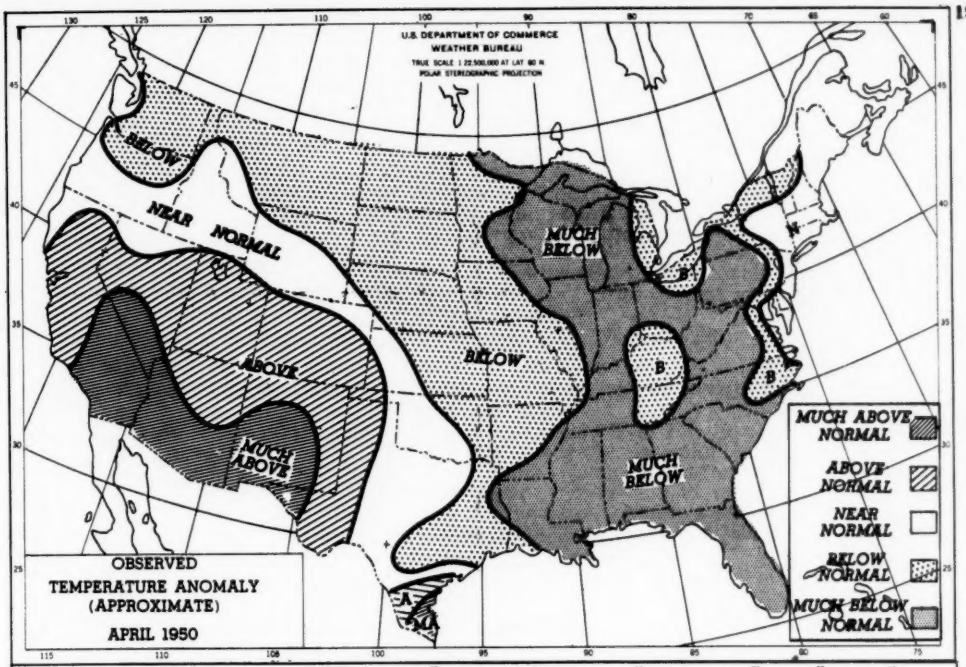


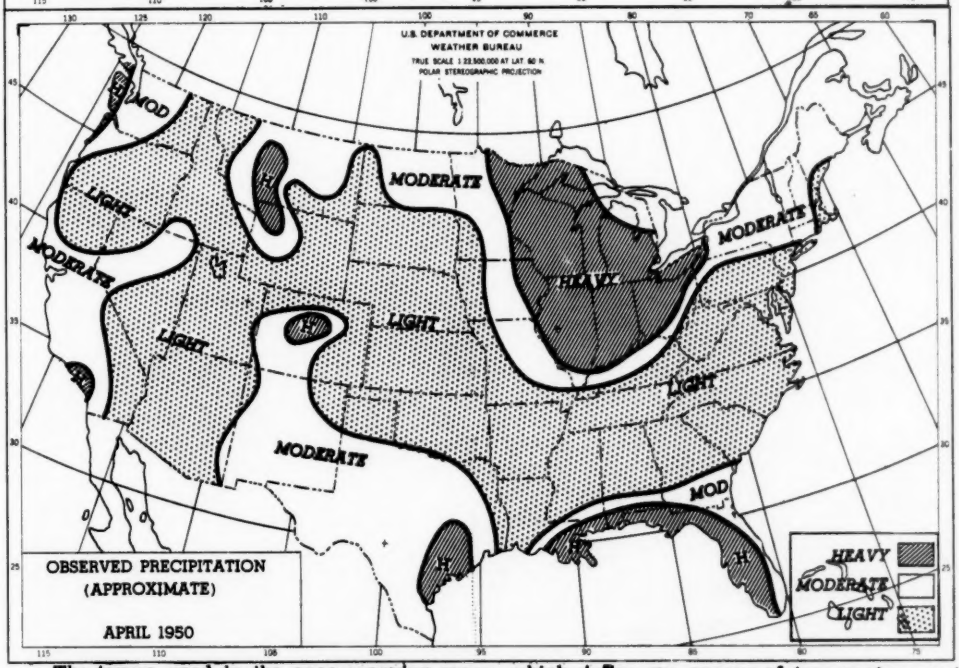


The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P.R.M.

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